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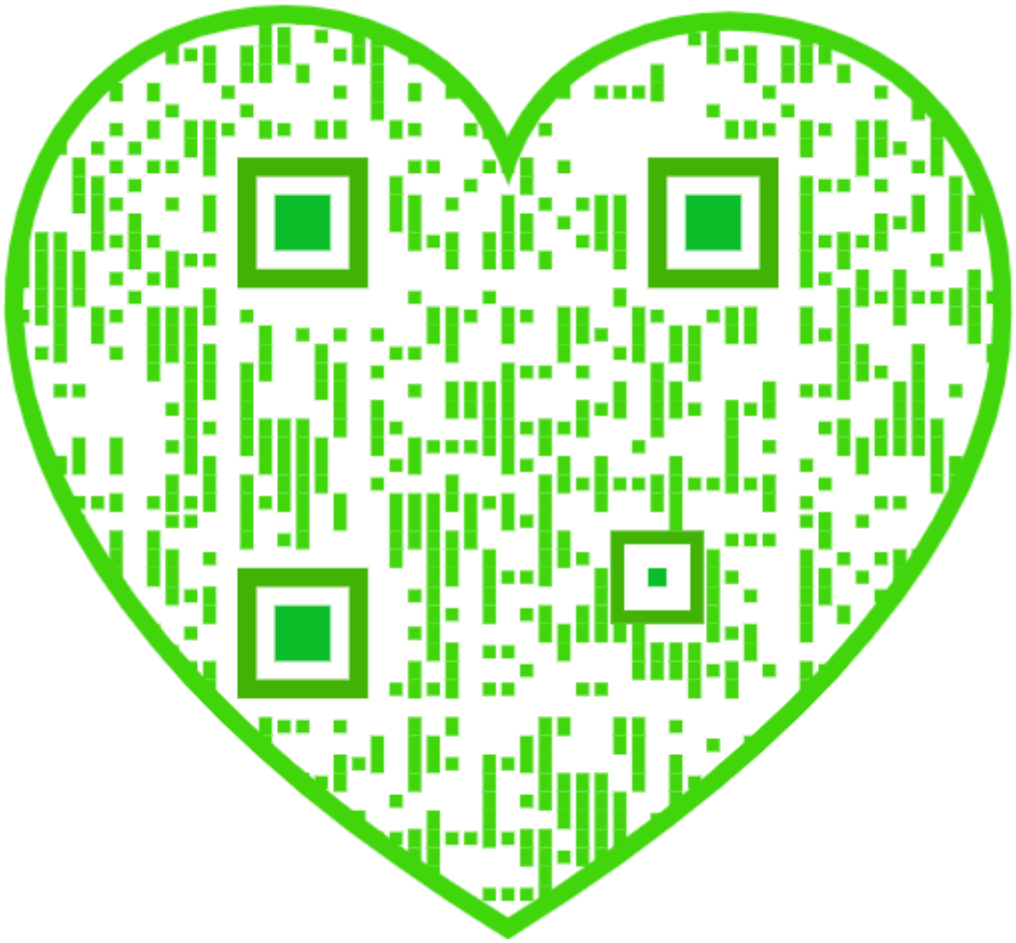

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INTERNAL MEDICINE

A WORK FOR THE PRACTICING PHYSICIAN
ON DIAGNOSIS AND TREATMENT
WITH A COMPLETE DESK INDEX

BY

NATHANIEL B. POTTER, M.D., JAMES C. WILSON, M.D.

IN THREE VOLUMES

ILLUSTRATED WITH 423 TEXT
ILLUSTRATIONS AND 14 IN COLOR

MEDICAL DIAGNOSIS

FIFTH EDITION REVISED AND ENLARGED IN TWO VOLUMES

Vol. II

CLINICAL APPLICATIONS;
NATURAL HISTORY OF DISEASES
DESCRIPTIVE MEDICINE

BY

JAMES C. WILSON, A.M., M.D.

Emeritus Professor of the Practice of Medicine and Clinical Medicine in the Jefferson Medical College and Physician to Its Hospital; Physician Emeritus to the Pennsylvania Hospital and Physician-in-Chief to the Lankenau Hospital, Philadelphia

ASSISTED BY

CREIGHTON H. TURNER, M.D.

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To

THE MEMORY OF MY

FATHER

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COLLABORATORS

J. Leslie Davis, M.D.
Norman B. Gwyn, M.D.
Frederick J. Kalteyer, M.D.
James Hendrie Lloyd, M.D.
Willis F. Manges, M.D.
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G. Canby Robinson, M.D.
William M. Sweet, M.D.
Corson White, M.D.
William Reynolds Wilson, M.D.

PREFACE TO VOLUME II

The term "Clinical Applications" needs no explanation. Its meaning is obvious. The use of the phrase "Natural History of Disease" is unusual and may need some comment. It means the sum of our knowledge of the origin, phenomena, course and termination of particular diseases and the changes revealed at autopsy in case of death. As these considerations have been the subject of Descriptive Medicine from the earliest times and have been little influenced by therapeutic measures until a recent period, they are in the main accurate. It is essential that the clinician should be thoroughly familiar with them.

Modern knowledge of causes has created Preventive Medicine and revolutionized Therapeutics, but the sagacious observations of the older physicians at the bedside are in no wise to be ignored.

I am under especial obligation to Doctor Byrom Bramwell for his courtesy in permitting me to increase the number of illustrations by the addition of several from his splendid Atlas of Clinical Medicine, reproduced in a size suitable for the present volume.

J. C. Wilson.

Philadelphia, January, 1920.

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A HANDBOOK
of
MEDICAL DIAGNOSIS

VOLUME II.

PART IV.

OF THE CLINICAL APPLICATIONS.
NATURAL HISTORY OF DISEASES—DESCRIPTIVE MEDICINE.

I.
DIAGNOSIS OF THE SPECIFIC INFECTIONS.

I. ENTERIC OR TYPHOID FEVER.

Definition. — An acute general infection caused by the Bacillus typhosus, characterized clinically by fever of prolonged duration, a scanty rose-colored, maculopapular eruption, enlargement of the spleen, abdominal tenderness, diarrhœa and tympanites, profound asthenia, and rapid wasting; anatomically by hyperplasia and ulceration of the lymph follicles of the intestines, enlargement of the mesenteric glands, and diffuse parenchymatous changes in the viscera.

Etiology. — Predisposing Influences. — Enteric fever is the prevalent febrile infection of the present historical epoch, just as typhus was that of the three hundred years preceding the beginning of the nineteenth century and the plague that of the Middle Ages. It owes its present wide distribution and great prevalence to faulty disposal of sewage and neglect of the simplest sanitary laws. Defective drainage and contaminated water and milk constitute the means by which the specific infecting organisms are distributed. Climate.—Enteric fever prevails in all parts of the world but is especially common in temperate climates. Season. — It is most common in the autumn months but occurs at all seasons of the year. Of personal causes: Sex.—Males and females are equally liable to the disease. The mode of life of the individual exposes males to the infection to a greater extent than females. In early childhood the sexes suffer alike: in late childhood and adolescence, boys more than girls. More men than women are admitted to hospitals. The conditions of camp life in military campaigns especially favor the spread of the infection. In the Spanish-American and South African wars enteric fever prevailed most disastrously. Age. — Enteric fever is especially a disease of youth and early adult life. The period of greatest liability is from the fifteenth to the twenty-fifth year. Exposure and immunity are to be considered in this connection. Exposure to the infection is probably greater after adolescence and the immunity acquired by the attack more general after the twenty-fifth year. Cases occasionally occur after sixty. Occupation and Social State.—

These conditions are without predisposing influence. Immunity.—Not all who are exposed contract the disease. An unrecognized or forgotten attack in childhood may have conferred immunity. The almost universal practice of immunizing vaccination against enteric fever among the armies of the present war has on the one hand so reduced the prevalence of that disease as compared with previous modern wars that its death rate has become practically negligible, while on the other hand the paratyphoid infections, not directly influenced by such vaccinations, have come into unsuspected prominence and importance. The necessity of controlling the prevalence of the latter by specific prophylactic vaccination became at once apparent. Immunization with each bacillus separately by means of a succession of inoculations has been superseded by the use of a mixed triple vaccine in which the three microörganisms are inoculated simultaneously, a method which is followed by an immunity against each of the constituent organisms as effective as that secured against any one of the three, employed alone for the first immunizing vaccination. This method of conferring immunity against the typhoid infection should be generally practised in civil life. There are families which show in successive generations an especial susceptibility. The immunity acquired by the attack is usually life-long. Second attacks have occurred in the course of several months or years and three attacks in the same person have been noted. In this connection the paratyphoid fevers are to be considered..

The Exciting Cause. — Bacillus Typhosus.—This organism, the bacillus of Eberth, is constantly present in enteric fever. It is a short, thick, flagellate, motile bacillus with rounded ends, growing readily on various culture media. It can now be differentiated from certain other bacteria, to which it bears a close morphological resemblance, especially the Bacillus coli communis, with which it is liable to be confounded. These organisms colonize with preference in the lymphoid tissues. They are distributed in recent infection in the solitary follicles and Peyer's patches of the intestines, in the mesenteric glands, spleen, bone-marrow, liver, and in the bile. They have been isolated from foci of suppuration in various parts of the body, from meningeal and pleural exudates and vegetations upon the endocardium. Their presence in the blood and rose spots may be demonstrated. In the course of the second week and afterward they have been isolated by culture methods from the stools. They are met with in the urine and sputum and have in a few instances been found in the sweat.

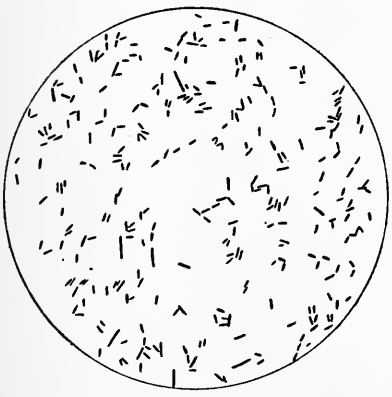


Fig. 206.—Bacillus typhosus.

Outside the body typhoid bacilli retain their vitality in water, snow, ice, the superficial layers of the soil, dust, and in fæces for periods varying from several days to many months. In milk they undergo rapid growth

without changing its appearance. They retain their vitality for three months in sour milk and for shorter periods in butter made from infected cream. The infecting principle is discharged from the body of the patient in the urine and especially in the faces. Its conveyance by means of water, milk, or other articles of food contaminated by such discharges in consequence of faulty sanitation is the source of the sporadic, endemic, and epidemic prevalence of the disease. Enteric fever is not contagious in the ordinary sense, when the introduction of bacilli-containing dejecta or secretions does not take place.

Infected water is the ordinary means of transmission. By this means the infection of milk, uncooked vegetables, salads, oysters, and clams occurs. Lobsters also may become infected when kept in cages in sewage-contaminated water awaiting a market. Natural ice, and vegetables grown upon soil fertilized by sewage, eaten raw, may convey the germs. Flies and atmospheric dust play an important part in the dissemination of the disease. The open latrines on the one hand and the unscreened mess table on the other were largely responsible for the terrible epidemics among our recruits in the practice camps at the time of the Spanish-American War.

When the bacilli find their way into the intestinal tract the evolution of the disease is as follows: Being resistant to dilute acids they are by no means wholly destroyed by the hydrochloric acid of the gastric secretion. If acid be absent or the water or other fluid containing them be ingested when hydrochloric acid is not secreted they pass into the intestine and colonize and multiply in the lymph structures, finding their way into the mesenteric glands and thence by means of the blood to the spleen, liver, kidneys, and bone-marrow. In these locations and elsewhere they form soluble toxins which, circulating in the blood, give rise to the fever and other constitutional symptoms.

Period of Incubation.—This is very uncertain. In some cases it appears to have been as short as four or five days or even less. It varies between two and three weeks. This period runs its course without symptoms, provided prodromes are not included.

Stage of Prodromes.—The onset is rarely abrupt. It is preceded by a period of impaired health characterized by malaise, feebleness, indisposition to bodily or mental effort, loss of appetite, headache, vertigo, and disturbed sleep. Abdominal uneasiness, even pain, and diarrhœa in the absence of laxatives are often present at this time. Slight but transient rises of temperature may occur; bleeding at the nose may occur.

The course of the attack varies between twenty-one and twenty-eight days. It may be divided into the stage of onset, the fastigium or fully developed disease, and the stage of decline upon which supervenes the convalescence. As there are anatomical changes in the lyr phoid structures of the intestines which run their successive courses in periods of five to seven days and correspond to changes in the symptom-complex of similar duration, it is convenient for purposes of description to divide the course of the attack into four periods of a week each.

Course of the Disease.—First Week.—The attack begins with a distinct elevation of temperature. This rise is frequently attended with chilliness, which may be repeated, but rarely by a pronounced chill. The patient

now in the majority of instances betakes himself to his bed. During the first four or five days the temperature rises in the evening from one to two degrees higher than upon the previous evening, and each morning a degree or more above that of the preceding morning. At the end of this time the temperature has reached its fastigium, 103°–105° F. (39.5°–40.5° C.), and with slight morning remissions remains, in the absence of complications, at this level until the end of the second week. During this period there are lassitude, headache, anorexia, thirst, a hot, dry skin, diminished urine, and restless sleep. The headache becomes progressively more severe and is attended with tinnitus aurium and delirium, at first mild and present usually upon waking. Epistaxis frequently occurs. It is usually slight but may be free. The tongue is coated and is seen to be of a bright red color at the edges and tip—"red tongue fever." At this time constipation is the rule but laxatives act with unusual energy. Toward the end of the first week spontaneous diarrhœa often occurs. There are cases, however, in which constipation continues throughout the attack. The spleen is enlarged; there is slight tympanitic distention of the abdomen and tenderness in the ileocæcal region. Gurgling upon pressure, often observed in this region, is without diagnostic value.

A few scattered, medium-sized, dry râles may usually be heard upon auscultation of the lungs. The pulse is rapid, 90–110, but less so in proportion to the rise of temperature than in many other acute diseases. It is full in volume, of low tension, and often dicrotic.

Second Week.—The fever now assumes the subcontinuous type, the range between the evening rises and morning remissions not greatly exceeding those of health. The symptoms become progressively more severe. The pulse is rapid and gradually loses its dicrotism. About the tenth day the headache ceases and is replaced by somnolence and stupor, which alternate with delirium usually wandering but sometimes noisy and active. The facies is dull, faintly flushed, sometimes slightly cyanotic. The lips and tongue are dry and sordes tend to collect upon the teeth and gums. The abdominal symptoms are aggravated; there is decided enlargement of the spleen, and between the sixth and tenth days the eruption makes its appearance. Traces of albumin are now to be found in many of the cases. The signs of bronchitis are more pronounced. Fine subcrepitant râles are heard at the bases posteriorly and slight dulness upon percussion may be found in this region.

Third Week.—The temperature shows morning remissions of increasing length. The pulse becomes more feeble and frequent, 110–140. The first sound of the heart is faint and may be inaudible. There is muscular tremor. Diarrhœa and tympanitic distention of the abdomen may increase or, if previously absent, now appear. There is often retention of urine and sometimes involuntary discharges of urine and fæces. Weakness is marked and wasting conspicuous. Stupor and delirium continue. The rash, which has appeared and faded in successive crops, now gradually diminishes. This is the period of severe complications, hypostatic pneumonia, bed-sores, parotitis, hemorrhages, and perforation.

Fourth Week.—The type of the fever is now intermittent, the morning remissions falling to normal or slightly subnormal ranges and the evening

rises progressively diminishing until they no longer transcend the normal The tongue becomes clean and moist, the diarrhœa ceases, and there is a desire for food. The spleen undergoes involution. The tympanites subsides. The pulse becomes stronger and fuller and the first sound and impulse of the heart more distinct. The convalescence may be postponed by the occurrence of various sequels or by relapse, and in some instances the symptoms of the fourth or even of the fifth week may continue to be the same which were present during the third—progressive asthenia, rapid and feeble pulse, abdominal distention, involuntary discharges, dry tongue, muttering delirium, stupor, and subsultus.

The foregoing sketch represents a severe attack unmodified by treatment and terminating in recovery. But from this typical picture of the

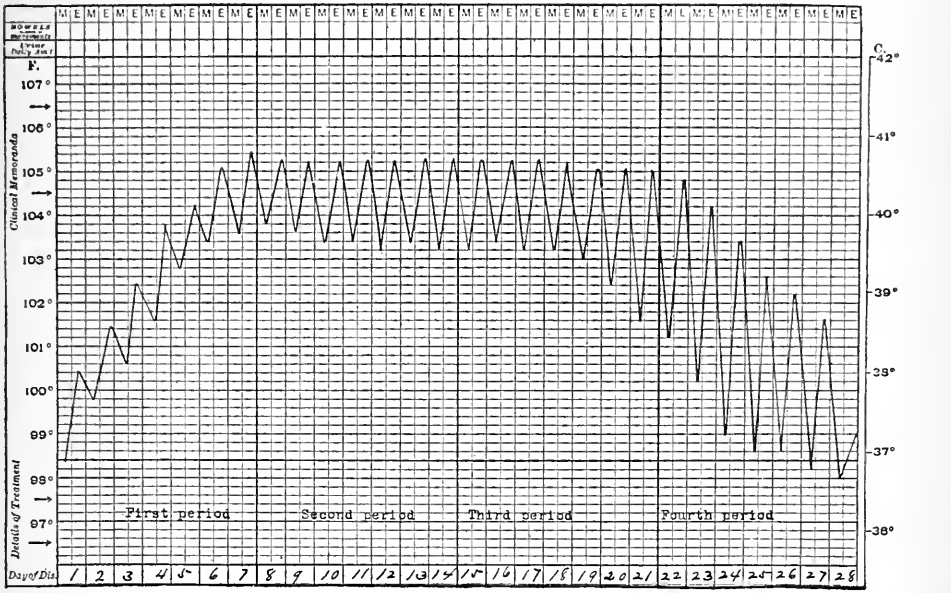


FIG. 207. —Temperature range in enteric fever.—Wunderlich.

disease there are many variations. The problem in diagnosis is to determine not the nature of a well-defined typical case of enteric fever—that any tyro in medicine can readily do—but to recognize the atypical cases and to differentiate them from the affections to which they present features of resemblance and to do this successfully at the earliest possible moment, since failure in this respect leads to indifference in regard to the search for the cause of the attack and neglect in the proper treatment and disposal of the evacuations.

Symptoms of Especial Importance in Diagnosis.—The association of certain symptoms is of cardinal importance in diagnosis: These symptoms relate to (a) the fever, (b) the pulse, (c) the rash, (d) the abdominal organs, and (e) the nervous system.

(a) **The Fever.**—1. REGULAR, SO-CALLED TYPICAL COURSE.—The temperature rises by regular step-like gradations, with marked evening exacerbations and slight morning remissions, until it reaches a range of

103°–105° F. (39.5°–40.5° C.) by the fourth or fifth day. From this period to the end of the second week its range is subcontinuous, the oscillations between the morning minima and the evening maxima but little exceeding those of health. During this time the temperature is scarcely modified by ordinary antipyretic measures and only gradually yields to systematic cold bathing. Toward the end of the second and throughout the third week the type becomes remittent with diurnal oscillations of gradually increasing length until in the fourth week the type is distinctly intermittent, the diurnal oscillations progressively diminishing at the expense of the evening rises until subnormal ranges are reached. The defervescence is by lysis. From this level the temperature again in the course of a few days rises to normal, but it remains for a time unstable and is liable to recrudescences of 1°–3° F., extending over a day or two, from the action of slight causes,—physical or mental effort, the visits of friends, constipation, the eating of solid food,—*febris carnis*.

This typical temperature range is much less common than it was formerly thought to be and numerous modifications occur in cases that in other respects must be regarded as typical.

2. Variations in the Temperature Range.—The fact that most of the cases do not come under observation until some days have elapsed and the temperature has attained its fastigium throws some obscurity upon the frequency of the gradual step-like ascent of the first week. In cases seen from the onset it is often absent and the temperature may reach 104°–105° F. in the course of twenty-four or forty-eight hours. This is especially common in the cases which begin abruptly with chills or in children with convulsions. Not rarely the temperature range is remittent throughout—infantile type, seen sometimes also in the adult. Not very rarely the temperature falls rapidly about the end of the second week,—defervescence by rapid lysis, or by crisis,—an event more common in the cases which begin abruptly than in those of gradual onset. Inverse temperature is observed very rarely in enteric fever and is without diagnostic significance other than that which arises from its occasional occurrence in tuberculosis.

The course of the temperature is sometimes interrupted by sudden falls. These declines may amount to 8° or 10° F. in the course of a few hours. They occur from hemorrhage from any cause and especially accompany intestinal hemorrhage. As a rule they are followed in the course of several hours, when the bleeding has ceased, by a rise to the former range, but the temperature often remains unstable; exceptionally the temperature stays low and the patient enters upon an early convalescence. Hyperpyrexia is not common in enteric fever. In very rare instances collapse may occur in the absence of hemorrhage or perforation.

Recrudescences of fever from trifling causes, occurring as a manifestation of the instability of the heat-regulating mechanism which follows the infection, are of no great importance. They are, however, to be differentiated from the symptomatic fever of an inflammatory complication, as pneumonia or pleurisy or venous thrombosis. In this connection the local phenomena and an increase of the leucocytes are of importance.

Subfebrile States in Convalescence.—In children and neurotic individuals there may be evening fever, 100.5°–102° F. (38°–39° C.), for weeks after

the symptoms of the disease have disappeared and convalescence is in other respects fully established. I have several times seen this condition in laboring men in hospital wards. It has been described as bed-fever. It disappears when the patient is allowed to sit up. A similar evening rise may be the sign of a latent complication.

The subnormal temperature of early convalescence is not important. It is especially liable to occur in feeble or greatly emaciated individuals but may be encountered in persons making a good recovery. In the course of a week or ten days the temperature rises to normal and regains its stability. The hypothermia of hemorrhage and the morning remissions of the later stages have been already described. That of cold bathing and other antipyretic treatment is transient and without diagnostic importance.

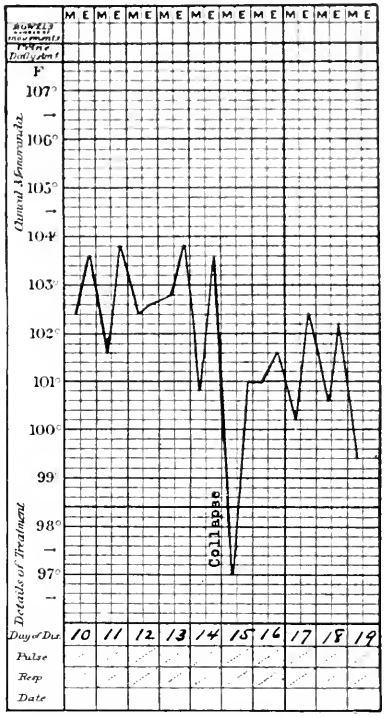


FIG. 208.—Collapse in enteric fever, 14th day. No hemorrhage. Ultimate recovery.

Relapses are characterized by a febrile range like that of the primary attack, except that the respective periods are shorter just as the relapse is shorter—ten days to two weeks. The gradual ascent, subcontinuous fastigium, and defervescence by lysis with remittent and intermittent curves are important from the standpoint of diagnosis.

Afebrile Typhoid. — Cases have been described in which the general constitutional symptoms and the duration of the case together with the eruption, enlargement of the spleen, dicrotism, and the diazo reaction have been present without fever or at most with only a trifling elevation of the evening temperature to subnormal ranges, and in which the etiology of the disease and the existence of a local epidemic have rendered the diagnosis of enteric fever in the highest degree probable. The diagnosis will become positive in the event of the occurrence of intestinal hemorrhage or upon post-mortem examination resulting in the demonstration of the characteristic intestinal lesions. In the absence of those events the diagnosis may then be made by the finding of the bacilli in the urine and stools or in blood cultures.

3. CHILLS.—Chilliness is not uncommon in the period of onset, but rigors are rare in enteric fever. Chills occasionally, however, occur with the initial rise of temperature; at irregular intervals during the course of the attack, followed by profuse sweating; sometimes upon the development of complications; after the administration of internal antipyretics, and in septic conditions. Chilliness and shivering are frequent at the end of tub-baths.

(b) **The Pulse.**—The characters and frequency of the pulse in relation to the fever are of diagnostic importance. The pulse is commonly full and of low tension in the beginning of the attack. Dicrotism appears early and is not only more common but also more marked than in any other acute disease. The frequency is increased but not proportionately to the elevation of the temperature. A pulse of 100–120 may within the second week be associated with a temperature of 104°–105° F. (40°–40.5° C.). Later the pulse becomes more frequent and feeble and its volume is much diminished. In grave cases with great prostration it may reach 160 and such a degree of enfeeblement as to be scarcely countable.

(c) **The Rash.**—The eruption occurs in at least four-fifths of the cases. It consists of lenticular, hyperæmic papules, slightly elevated above the surface of the skin, of a pale rose color, one to four mm. in diameter and disappearing upon pressure or when the skin is made tense. These roseolous maculopapules may be felt by the finger. They must not be confounded with the dense papules or small pustules of acne which are common upon the trunk, especially the back, of young persons. They appear in successive crops and vary in number from two or three, found only upon careful search, to a somewhat copious roseola distributed upon the surface of the trunk and extremities. Contrary to the general opinion, I am now of the belief that cases characterized by an abundant rash frequently run a severe course. The eruption first makes its appearance between the end of the first and the middle of the second week and commonly in the epigastric zone upon the anterior surface of the body. It is also frequently noted upon the back, between the shoulder-blades. It may in rare instances be seen upon the face, especially in young persons of fair skin. The spots are circular or oval with well-defined borders. They gradually fade in two or three days, leaving an area of pigmentation the degree of which varies according to the complexion of the individual. In the majority of cases no new crops appear after the defervescence begins, but exceptionally the spots continue to appear after the temperature has fallen to normal.

Other points of value in diagnosis in connection with the skin are the out-cropping of sudamina as the fever begins to decline; the occasional occurrence of purpura; an infrequent erythematous eruption at the outset, resembling that of scarlet fever; a fine branny desquamation in children; the presence of the *tache cérébrale*, and the great infrequency of herpes labialis in comparison with malaria, croupous pneumonia, and cerebrospinal fever.

(d) **The Symptoms Relating to the Abdominal Organs.**—Splenic enlargement may be made out upon palpation, the border of the organ extending below the ribs, especially on deep inspiration. The results of percussion are obscured by the meteorism which is common after the beginning of the second week. A splenic tumor is demonstrable in more than eighty per cent. of the cases.

Diarrhœa is a variable symptom. It is present at some time in the course of the majority of the cases, often alternating with constipation. There are, however, epidemics in which constipation throughout is the rule. Diarrhœa is more common in the later course of the attack. It is caused by the associated catarrh rather than by the ulcers and is indicative of extensive rather than of deep ulceration. The number of the stools varies

from two or three to eight or ten in twenty-four hours. They are usually large, thin, grayish-yellow in color and of a granular composition. They very often contain one or more soft scybalous masses of the size of a walnut. The reaction is alkaline and the odor foul. On standing, the fluid and solid constituents separate into two layers, the upper containing albumin, salts, and coloring matter, the lower epithelial débris, cellular elements, fat crystals, triple phosphates, and later in the disease sloughs from the necrotic Peyer's patches, and microscopic blood. In many cases the bacilli of Eberth may be found after the middle of the second week. This separation of the stools into layers is not often seen in other forms of diarrhœa, but cannot be regarded as pathognomonic. In truth it cannot be said that the stools of enteric fever are characteristic in any diagnostic sense. The familiar comparison with pea-soup is inexact and misleading and might well be discarded with a multitude of other unscientific, traditional, false phrases from the language of descriptive medicine.

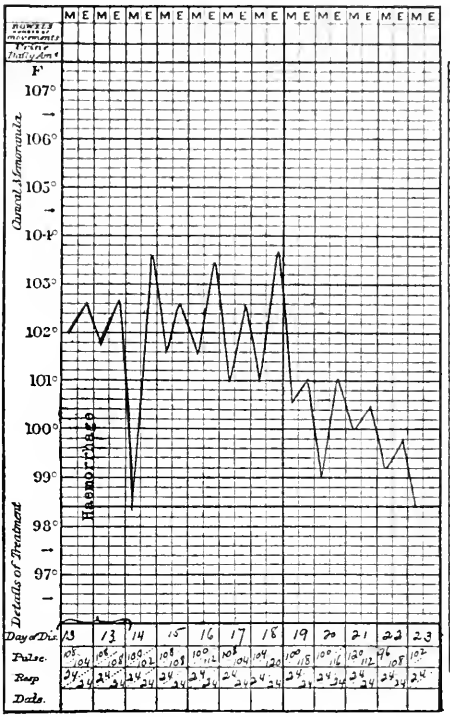


Fig. 209.—Enteric Fever. Hemorrhage on the 13th day; recovery. Woman, aged 23.

Tenderness and Pain.—Tenderness in the ileocæcal region is common in the early course of the attack and important in the differential diagnosis between enteric fever and appendicitis. The tenderness is in some instances confined to the umbilical region; less often it may be elicited upon pressure over almost any part of the abdomen. Tenderness and pain in the abdomen may be symptomatic of an over-distended bladder, pleurisy, crural phlebitis, or other acute affection. In a large proportion of the cases no definite cause for these symptoms can be discovered. Pain and tenderness are occasionally associated with intestinal hemorrhage but are present and intense in almost all cases of perforation.

Intestinal Hemorrhage.—Hemorrhage from the bowel is a serious accident, occurring in from three to ten per cent. of all cases. It varies in amount from a trace of blood in the stools to a profuse and fatal blood loss. Large hemorrhages most commonly occur about the time of the separation of the sloughs, namely, between the close of the second and the beginning of the fourth week. The slighter hemorrhages which take place earlier than this period are due to oozing from the hyperæmic Peyer's

patches; those which occur after it are to be ascribed to the mechanical disturbance of unhealed ulcers by the peristaltic movement of the bowel and the contact of the intestinal contents. Intestinal hemorrhage usually comes on without premonitory symptoms. It may reveal itself at once by the discharge of blood from the anus, with faintness, feeble pulse, pallor, and a rapid fall of temperature to the extent of several degrees, or by collapse symptoms which may terminate in death before the blood appears in the stools—concealed hemorrhage. Intestinal bleeding may be symptomatic of a general hemorrhagic condition manifested also by petechiæ and hæmaturia or oozing from other mucous surfaces.

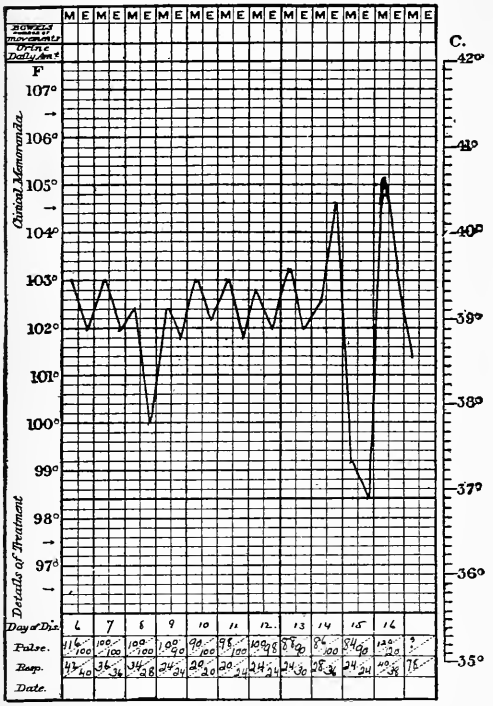


FIG. 210.—Enteric fever. Signs of perforation, 14th day; operation refused; death two days later.

PERFORATION. — Intestinal perforation is the gravest accident that occurs in enteric fever. Its frequency is about three per cent. of all cases. It may happen in otherwise apparently mild cases but is more common in severe cases in which diarrhœa and meteorism are marked or in which hemorrhage has occurred. Nearly fifty per cent. of the cases occur in the third or fourth week. The symptoms are first those caused by the perforation itself and the escape of the contents of the bowel into the peritoneum, and second, those of the resulting peritonitis. Of the first group, sudden, sharp pain in the right lower quadrant of the abdomen, increasing in severity, attended by general or local tenderness, is the most significant. Next in order of importance is rigidity of the abdominal muscles, which become spastic upon palpation. Irritability of the bladder and frequent micturition are not uncommon. Much less frequent are collapse symptoms, fall in temperature, increase in pulse-frequency, and coldness of the surface with sweating. In grave toxæmia with stupor the symptoms of perforation may be obscured.

The second group comprises the symptoms of the consecutive peritonitis. The local symptoms are of great importance since the life of the patient may depend upon their early recognition. They consist of muscular rigidity with pain upon pressure and deep breathing, limitation of the respiratory movements, obliteration of the lower border of the liver and splenic dulness from the presence of free air in the peritoneal cavity, fulness and

tenderness upon digital examination per rectum; absence of the peristaltic murmur upon auscultation, friction sounds in the upper hepatic area or elsewhere, and in the majority of the cases a rapidly progressive leucocytosis. These phenomena are not all present in every case but the presence of several of them justifies the diagnosis of typhoid peritonitis in its early stage. Their progressive development is soon followed by the occurrence of more ominous signs. The spastic contraction of the muscles of the abdominal wall is replaced by tympanitic distention which progressively increases. Percussion in the flanks reveals flatness in the most dependent region, the sign of an accumulating exudate. The pain is less urgent; the tenderness less acute. Further displacement of the liver and splenic dulness in an upward direction is noted and the impulse of the heart may be found as high as the fourth interspace. In a small number of the cases perforation and peritonitis occur with a flat or even retracted abdomen.

The general symptoms indicate the gravity of the condition. They consist of pallor, an expression indicative of pain, which develops later into the facies Hippocratica, profuse clammy sweating, a feeble, thready, frequent pulse, hiccough, and vomiting. The respiration is shallow, the heart sounds indistinct, the temperature, which may have fallen upon the occurrence of perforation, rises again, and the kidneys secrete little or no urine. This formidable array of symptoms, the forerunners of the approaching fatal issue, may be masked by those of an antecedent overwhelming toxæmia.

Perforative peritonitis without the escape of air into the peritoneal sac may arise from the rupture of a pseudo-abscess, softened mesenteric gland, or a splenic or hepatic abscess. A general or local peritonitis may occur in consequence of infection through the thinned base of a deep ulcer without rupture.

(e) **The Nervous System.**—There is nothing characteristic in the nervous symptoms of enteric fever; yet their association with the phenomena mentioned in the foregoing pages constitutes a symptom-complex that is in the highest degree significant.

Headache, sleeplessness, and a condition of the nervous system which renders physical and mental effort alike difficult characterize the period of prodromes. The headache may involve any part of the head or be general. Its most constant peculiarity is that it becomes worse toward night. It usually ceases spontaneously about the middle of the second week and is replaced by somnolence and stupor—a change which is of diagnostic value. Tinnitus aurium frequently accompanies the headache and deafness develops with the progress of the disease.

Delirium sometimes begins early and is usually of mild character. The patient can be roused, his attention fixed and his replies become apparently rational. The delirium is at first nocturnal. Later it becomes continuous and marked. It may not appear until the second or even the third week. In other cases it is noisy and restless and attended with efforts to get out of bed. In hard drinkers it may be associated with tremor and the hallucinations peculiar to delirium tremens. Abrupt changes in the form of delirium occur. The patient who has been apathetic and wandering may without warning develop an active and purposeful delirium with suicidal tendencies. The enteric fever patient who has become delirious

must under no circumstances be left alone. Somnolence and hebetude are common and in the severe cases these symptoms deepen into a stupor from which the patient cannot be roused. Coma vigil, tremor, subsultus tendinum, and carphologia are among the most ominous symptoms of the disease.

Convulsions are rare. They may occur in children at the onset. In the course of the attack and especially in adults they may be hysterical, uræmic or symptomatic of some nervous complication, as encephalitis or thrombosis of cerebral arteries or veins.

Varieties.—Enteric fever is in the strictest sense a disease of the whole organism. Scarcely an organ escapes; not a function goes on normally. The duration of the attack with profound derangements of nutrition exposes the defenceless tissues to the secondary invasions of pathogenic organisms both local and general. In different cases various organs bear the brunt of the attack according to the personal predisposition of the individual. Variations in the intensity of the infection,—virulence of the bacilli,—the resistance of the individual,—integrity of the tissues, degrees of hereditary or acquired immunity or predisposition,—complications, sequels, relapse, the management of the case and the surroundings likewise modify the severity of the attack. Hence a most diverse and complex symptomatology. A full account of enteric fever in all its relations would constitute an epitome of the Practice of Medicine. It is important to remember that the disease presents the widest variations from a typical course alike in its mode of onset, its intensity, the prominence of certain symptoms, and in its duration. Errors in diagnosis in doubtful cases are to be avoided only by the routine employment of every resource of clinical medicine with due regard to the teachings of the clinical laboratory and the post-mortem room. From the standpoint of the infection the following varieties may be recognized:

(a) **Ordinary Form with Well-developed Intestinal Lesions.**—This group includes the great majority of the cases. The lesions of the lymph structures are well marked and more or less extensive; the mesenteric glands and spleen are enlarged, parenchymatous changes are present in the viscera. The anatomical diagnosis can be made in the absence of a history of the symptoms.

(b) **Cases Characterized by Slight Intestinal Lesions.**—The changes in the lymphoid structures of the intestines may be superficial and limited in extent and therefore readily overlooked, or if death has occurred late in the attack the ulceration may have already healed. The symptom-complex may be that of an ordinary attack with or without mild intestinal symptoms, of a general sepsis with high fever and marked nervous symptoms,—so-called typhoid state—typhoid septicæmia,—or of an affection of one or more viscera with profound constitutional disturbance. The organs especially involved may be the liver, gall-bladder, lungs, pleura, kidneys, endocardium, or meninges. It is in the last group that the cases are found which are described as pneumotyphus, in which the attack sets in with pulmonary symptoms; pleurotyphus, beginning with an acute pleurisy; nephrotyphus, in which the general symptoms and urinary findings of an acute nephritis are present at the onset, and the cerebrospinal form in

which the attack begins suddenly with urgent symptoms of disturbance of the nervous system. In all of these unusual forms the symptoms which dominate the clinical picture at the onset shortly, and mostly in the course of the first week, become subordinate to those characteristic of enteric fever and the attack generally runs its subsequent course in the usual way and time. In many of these cases, however, the intestinal lesions are well developed. To elevate these groups of cases into separate varieties is to increase the difficulties of the student and teacher alike without any compensatory advantages of classification.

(c) **Cases Characterized by the Absence of Intestinal Lesions.**—In some of the cases the true nature of the affection has not been demonstrated; in others lesions such as those of tuberculous ulceration, by which the bacilli of Eberth may have found access, were present. There remain, however, a limited number of cases in which the Bacillus typhosus has been demonstrated in the organs, the symptoms have been characteristic, and death has occurred at a time when the lesions of the gut are commonly conspicuous, yet none have been discovered. The possibility that the bacilli have found entrance by way of the intestinal wall without giving rise to demonstrable lesions has been suggested. Infection by way of the respiratory passages has not been demonstrated. Even in the cases of pneumotyphus the absence of intestinal lesions to which the early lung affection may have been consecutive has not been established.

(d) **Mixed or Secondary Infections.**—The conditions caused by the Bacillus typhosus impair the powers of resistance. A secondary invasion of colon bacilli, streptococci, staphylococci, or the pneumococci may occur with the development of consecutive local and constitutional phenomena. This true mixed infection may take place in any disease and is to be discriminated from other specific infections which occur as complications or intercurrent affections, as infection with Bacillus tuberculosis, Bacillus diphtheriæ, Streptococcus Fehleisen or, the malarial parasite.

(e) **Cases Presenting the Symptoms of Enteric Fever but due to other Organisms—Paratyphoid.**—Researches conducted since 1896 have shown that a symptom-complex not to be distinguished from enteric fever may be caused by organisms other than the Bacillus typhosus, which stand in their cultural and agglutinating properties between B. typhosus and B. coli communis, and that B. coli may perhaps play the same etiologic rôle. This fact does not, however, impair the universal belief in the specific nature of B. typhosus and enteric fever.

As regards intensity the following forms may be described:

(a) **The Mild Form—Typhus Levissimus.**—The fever is moderate, not exceeding 102°–103° F. (39°–39.5° C.) in the evening. The symptoms characteristic of the ordinary form are present but are of mild intensity. Headache, weakness, epistaxis, rose spots, and the signs of splenic enlargement are present, but the illness is so slight that it is difficult to make the patient realize its true nature. Diarrhœa is not common. These cases are often regarded as simple continued fever, febricula, or gastric fever. Their duration varies from eight to fourteen days.

(b) **The Abortive Form.**—The onset is abrupt and marked by shivering or a chill. The temperature rises abruptly and may reach 104° F.

(40° C.). Rose spots appear early, often before the fifth day. At the end of the first week, or early in the second, the temperature falls by rapid lysis or even by crisis with profuse sweating and the patient enters upon convalescence. These cases are sometimes seen in epidemics. The recognition of the true nature of the mild and abortive cases is of the utmost importance from the standpoint of prophylaxis.

(c) **The Latent or Ambulatory Form—Walking Typhoid.**—The symptoms are slight and the patient continues to attend to his affairs as usual. There is feverishness and a feeling of illness. Diarrhœa is commonly present but not urgent. The rose spots and enlarged spleen are often found in the routine examination of walking typhoid patients; or sudden delirium, hemorrhage, or perforation may occur. Cases belonging to this group are more common in men than in women and among laboring men, tramps, and others who habitually give little attention to their subjective symptoms. They are also encountered with some frequency among school-boys.

(d) **The Grave Form.**—The symptoms may at first be of moderate intensity. More commonly they are severe from the onset. The infection is intense. The temperature is high, 105°–106° F. (40.5°–41° C.), with very trifling remissions, and the duration of the fever may be protracted into the fifth or sixth week. To this category must be assigned the cases of mixed or secondary infection and the cases beginning with severe symptoms referable to the lungs, kidneys, and nervous system.

Modifications of the course of the attack as determined by anatomical and physiological conditions peculiar to the individual give rise to the following forms:

(a) **Enteric Fever in Children.**—This disease is not common in infancy. The nature of the food and doubtless the presence of an immunizing substance in the milk of the mother protects sucklings. Cases have, however, been reported in the first week of life and occasionally in the first year. Enteric fever is not at all uncommon after the second year. The onset may be insidious; commonly it is abrupt with high temperature. The type of the fever in a majority of the cases is remittent throughout—infantile remittent of the early writers. Nose-bleeding and diarrhœa are comparatively infrequent but bronchial catarrh begins early and is often moderately severe. There is nothing peculiar about the rash, which may be sparse or plentiful. Tympanites is commonly slight and intestinal hemorrhage and perforation much less common than in adults. Nervous symptoms are often prominent. The attack may begin with convulsions. Drowsiness alternating with insomnia, and mild delirium interrupted by sudden outcries and spells of fretfulness are observed. Aphasia, usually transient, and noma are prominent sequels. The mortality is much lower among children than in older persons. The marked differences in the course of the disease in childhood and after puberty, and especially the very common occurrence of fever of remittent course in connection with the symptom-complex just described, warrant the division of the cases of enteric fever into two great groups, those of the Infantile and those of the Adult Type. Those of the infantile type are milder than those of the adult type and the prognosis is more favorable. Cases of the former sometimes occur among adults; of the latter among children. The prog-

nosis is less favorable in an attack of adult type in a child; more so when the infantile type occurs in later life. This is in accordance with a long recognized fact, namely, that in the absence of complications the prognosis is more favorable in proportion as the morning remissions are longer, *i.e.*, as the temperature curve conforms to the remittent type of fever. It has been found also that treatment which systematically brings about large oscillations between the morning and evening temperatures, as the cold bath treatment, also renders the prognosis more favorable.

(b) **Enteric Fever in the Aged.**—The course of the disease is much modified when it occurs in middle life or in elderly persons. The temperature range is irregular and not so high. The rose rash and splenic tumor are often absent. Diarrhœa and tympany are often troublesome and there is a marked tendency to complications, especially those affecting the respiratory tract—pneumonia, bronchitis.

(c) **Enteric Fever in Pregnancy.**—The pregnant woman enjoys no immunity against the disease. The fever may develop at any time, but is more commonly met with in the first half of pregnancy. Abortion or premature labor occurs in a large proportion of the cases. The maternal mortality is high—sixteen to twenty per cent. Infection of the fœtus does not always follow, but when it occurs the child dies either in utero or shortly after delivery. Recent investigations have shown that the bacilli may pass by way of the placenta to the child and cause a typhoid septicæmia without intestinal lesions. The positive Widal reaction has been observed with fetal blood.

Complications and Sequels.—Complications and sequels are more common in enteric fever than in any other acute infectious disease. A recognition of this fact is of great importance in diagnosis, since cases occur in which the prominence of a complication may mask the symptoms of the primary disease.

The following more important complications are to be considered:

(a) **Complications Involving the Digestive and Abdominal Organs.**—Ulcerative stomatitis occasionally occurs. Phlegmonous and pseudomembranous angina is a rare complication, which may develop in the third week and usually proves fatal. Parotid bubo, usually single, sometimes double, is a grave but not necessarily fatal complication in severe cases. It may be followed by extensive sloughing or by angina Ludovici, venous thrombosis or pyæmia. Hæmatemesis is of extremely rare occurrence in enteric fever. It may result from the specific lesions implicating agminate follicles present in the gastric mucous membrane or from a peptic ulcer.

The enlargement of the spleen may attain such a degree that the capsule may burst. Rupture of this organ is more likely to be the result of abscess formation following infarct. The latter condition owes its occurrence to embolism or venous thrombosis.

The liver itself is rarely the seat of changes which attract attention. Jaundice is of very infrequent occurrence. Hepatic abscess is exceedingly rare. Cholecystitis is, on the other hand, common. Pain, tenderness, and muscular rigidity in the region of the gall-bladder may be noted in most of the cases. Distention of the viscus—gall-bladder tumor—may be recognized upon nice palpation and percussion. Perforation may occur with

the symptoms of intestinal perforation—extreme pain, tenderness, rigidity, fall of temperature, collapse symptoms, and the general and local signs of peritonitis. A suppurative cholangitis may occur. More commonly the symptoms gradually subside and recovery follows. There may, however, be remote consequences. The bacilli frequently give rise to chronic cholecystitis with recurrent paroxysms and to cholelithiasis.

Persistence of Bacillus typhosus After Recovery.—In by far the greater number of cases inflammation of the bile-ducts and gall-bladder terminates in recovery, but in about 2 per cent. it persists, especially in the gall-bladder, and the bacillus continues to multiply in the latter for an indefinite period. From this viscus it passes from time to time in considerable quantities into the gut and may be recovered from the fæces. These cases constitute a group of individuals now known as "typhoid carriers" who are a constant source of danger to the public, since they may spread the infection without giving rise to suspicion. Many of the sporadic cases, the origin of which has been involved in obscurity, are now attributed to these "carriers." Many of them are women and the subjects of cholelithiasis. The urine for several weeks may be in the absence of symptoms laden with virulent typhoid bacilli.

Intestinal hemorrhage and perforation have already been considered. These events are so directly due to the specific lesions of enteric fever, they occur with such frequency, and require such a degree of importance in the consideration of the subject that it seems more in accordance with the facts to regard them not as complications, but rather as accidents in the disease.

(b) **Complications Affecting the Respiratory Organs.**—Laryngeal ulceration is common in the severe cases. It may consist merely of superficial erosion and run its course without symptoms. It may, on the other hand, give rise to hoarseness, pain and difficulty in deglutition. Finally, it may produce perichondritis, in the course of which œdema of the glottis may occur. Bronchitis is prominent in infancy and often severe in old persons. Hypostatic pneumonia and deglutition pneumonia are almost always present in severe cases after the middle of the second week. Pulmonary œdema is a terminal condition.

Lobar pneumonia occurs, (1) as an initial condition—pneumotyphus. The onset is abrupt with chill, high temperature, pain in the side. Cough and bloody sputa occur. After a day or two the signs of consolidation occur and the case presents the complete clinical picture of an ordinary croupous pneumonia. Crisis does not occur and by the end of the first or the middle of the second week rose spots appear and the symptoms of enteric fever are unmistakable. In the absence of rose spots, the uncertainty as to whether the case is one of croupous pneumonia with so-called typhoid symptoms resulting from secondary infection or enteric fever with early pulmonary localization can only be cleared up by a bacteriological diagnosis—presence or absence of Eberth's bacilli in the sputum, urine, and pus, blood culture, Gruber-Widal test. (2) Croupous pneumonia is a common and serious complication—intercurrent disease—in the second or third week. It usually occurs in cases already otherwise severe. The

symptoms are not usually well developed. Cough is slight, rusty sputum may be absent, and the presence of the pulmonary consolidation may only be discovered upon routine examination. Secondary gangrene of the lung may develop. Pulmonary gangrene in enteric fever is more frequently the result of the breaking down of an infarct. Abscess of the lung constitutes one of the less common complications.

Pleurisy is by no means rare. It may be fibrinous, serofibrinous, or purulent. The effusion is often small and circumscribed. It may follow the signs of an infarct—local pain and dulness, increased fever, and hemorrhagic sputa. Typhoid bacilli have frequently been found in both the serous and purulent pleural exudates.

Pulmonary tuberculosis is not rarely an associated affection. The patient may be already phthisical, in which case the enteric fever plays the rôle of an intercurrent disease, or a latent tuberculous process may become active. Hæmoptysis may be profuse, even fatal. Pneumothorax has been observed. It may result from the rupture of a peripheral abscess of the lung.

(c) **Complications Affecting the Circulatory Organs.**—Pericarditis is exceedingly uncommon. It has been observed in children and in connection with pneumonia. Endocarditis is likewise rare.

Myocardial changes are on the other hand most frequent. They begin early and are often well established by the end of the second week. The heart is soft, flabby, and of a pale yellowish—faded-leaf—color. Upon the table it often flattens into a formless mass. Microscopically the changes are those of parenchymatous degeneration and interstitial myocarditis. There may be fatty degeneration of high grade. Feebleness of the pulse, faint, even inaudible first sound, profound asthenia, fatal collapse may be the manifestations of the changes in the myocardium. Acute dilatation with relative insufficiency, thrombus formation and visceral engorgements likewise arise and are attended with their usual symptoms and signs. There is a direct relationship in patients who recover between the myocardial changes of enteric fever and chronic myocarditis, the symptoms of which develop later in life, as can be learned from the anamnesis.

Vascular occlusions occur both in the veins and arteries. They may result from embolism or thrombosis. Obliteration of the femoral artery may occur with gangrene of the foot and leg. Obliteration of both femorals with extension of the clot into the aorta has been observed. The condition has been ascribed to local arteritis with thrombus formation.

Venous thrombosis is of comparatively frequent occurrence—two to four per cent. of the cases. It is usually unilateral, sometimes bilateral, the left side being first affected, the right later. In far the greater number of cases the femoral vein is the seat of the occlusion; less frequently the popliteal or the long saphenous. The clot may extend along the vein from the point of formation. The greater liability of the left femoral vein has been attributed to the relative retardation of the blood-flow in the left common iliac vein caused by the pressure of the right iliac artery which crosses it. The occurrence of venous thrombosis is attended by elevation of temperature, pain, tenderness, enlargement, and tense œdema of the leg. It may come on later in the attack or not until after defervescence. Eberth's

bacilli have been found in the clot and in the wall of the vein. Suppuration and pyæmia may occur. A fragment of the clot swept into the blood stream may cause sudden death by plugging of the pulmonary artery. Gangrene does not result from venous thrombosis. As the collateral circulation is established the enlargement of the leg subsides, but many patients are obliged to wear an elastic stocking for months or even years.

Thrombosis of the cerebral sinuses is a rare accident. Infarction of the lungs, spleen, or kidneys occurs as the result of arterial occlusion, due more commonly to thrombosis than embolism.

Gangrene as a sequel of enteric fever most commonly affects the feet and legs. Gangrenous areas occur less frequently upon the face, neck, and trunk. The genitalia, especially in girls, the nose and ears may also be affected.

The blood undergoes important changes. In the third week the erythrocytes and hæmoglobin are reduced. A gradual increase to normal takes place during convalescence, the hæmoglobin, which has suffered a relatively greater reduction than the corpuscles, regaining the normal more slowly than the latter. These changes are without diagnostic value.

Very important, however, is the fact that there is a reduction of the leucocytes during the whole course of the attack—leucopenia. This condition is of actual diagnostic value in the differentiation between enteric fever and septic states, and other infectious diseases which resemble it more or less closely. A leucocytosis occurs when in the course of enteric fever a local inflammation arises or pneumonia or some other affection characterized by an increase in the number of the leucocytes occurs as an intercurrent disease. The large mononuclear and transitional forms are increased and the polynuclear neutrophiles are greatly diminished. In contrast to these changes, the polynuclear neutrophiles are increased in inflammatory conditions, as in abscess formation or perforation, a fact of diagnostic value.

(d) **Complications Affecting the Nervous System.**—The nervous system, as has been pointed out above, in all cases manifests to a greater or less degree the effects of the intoxication. These effects are apparent at the onset of the attack and vary in intensity from the headache and indisposition to mental effort seen in the mildest cases to the furious symptoms of meningitis. The latter cases, which fortunately are extremely rare, are characterized by intense headache, photophobia, painful retraction of the muscles of the back of the neck, muscular twitchings, rigidity, and in some cases convulsions. The onset is abrupt and vomiting may occur. At the end of a week the symptoms become less intense, in a considerable proportion of the cases almost as suddenly as they appeared. The headache ceases, rose spots appear, there is palpable enlargement of the spleen, and the case presents the clinical features of an ordinary attack of enteric fever which runs the usual course. These are the cases described as cerebrospinal typhoid. In fatal cases of this group the lesions of meningitis are not found.

Inflammation of the meninges of the brain has been observed. It is an extremely rare complication. Typhoid bacilli have been isolated from the exudate in pure culture. Kernig's sign was present in a case of enteric

fever with meningeal symptoms recently observed. Lumbar puncture may be necessary in the differential diagnosis between this form of enteric fever and cerebrospinal fever.

The rare cases of thrombosis of the cerebral veins and sinuses are characterized by local and general convulsive movements, active delirium, and rapidly developing coma.

Neuritis is an infrequent complication. It is usually confined to a single nerve; sometimes it is symmetrical. Less frequently there is a general peripheral neuritis. Neuritis affecting a single nerve area may develop during the course of the attack, or, as is more commonly the case, after convalescence has begun. There is severe pain in the affected limb with exquisite tenderness over the trunk of the nerve. There may be swelling and redness. The extensors are more commonly involved and wrist-drop or foot-drop may occur.

Tender toes constitute the manifestation of a form of neuritis not very uncommon. This distressing but not very serious condition may occur under any treatment but is said to be more frequent in cases treated by systematic cold bathing. The tips of the toes, their pads, and sometimes the pads at their bases are painful and exquisitely sensitive so that it is impossible for the patient to bear the weight of the bedclothes. There is neither redness nor swelling, and the condition—which begins toward the end of the attack—usually disappears in the course of a week or ten days.

Multiple neuritis, sometimes giving rise to paraplegia, palsies of individual nerve-trunks from neuritis and poliomyelitis, hemiplegia from thrombosis or meningo-encephalitis, and tetany may develop during convalescence. These sequels are of extremely rare occurrence and of secondary importance in the diagnosis.

Postfebrile insanity is encountered more frequently after enteric fever than any other acute infection. It belongs to the group of confusional insanities and is the manifestation of profound nutritional derangements in neurotic individuals. It has occurred in my experience in cases in which active delirium has been present during the attack. There are persistent hallucinations, melancholia, and speech derangements. The prognosis is good. A majority of the cases recover in the course of six or eight months. After that period the outlook becomes progressively less favorable, though I have seen a case recover at the end of a year.

Complications relating to the eye are exceedingly rare. Of those affecting the ear, otitis media is common. There have been years in my service in the Pennsylvania Hospital in which no cases of this complication have occurred, and years in which it has been extremely frequent. The otitis commonly develops insidiously but it sometimes comes on with a chill and increase of fever. The ears should be examined as a matter of routine. Mastoid disease may occur.

(e) **Other Complications.**—Febrile—toxic—albuminuria is of very frequent occurrence as in the other serious infectious diseases. Hyaline and granulohyaline casts may be found in the centrifugated urine. This condition passes away with convalescence. Eberth's bacilli are demonstrable in the urine in a large proportion of the cases. In some cases they have been found in the urine of persons months, even years, after the attack.

The attack may begin with the symptoms of an acute nephritis—nephrotyphus. The toxic albuminuria occasionally leads to the development of an acute nephritis with scanty urine of high specific gravity, a large amount of albumin, and microscopical blood. This complication may be the cause of a fatal termination of the case. Otherwise it gradually subsides and complete recovery ensues. Polyuria during the course of the attack is an extremely rare complication. Chronic nephritis is a rare sequel. The multiple foci of round-celled infiltration—lymphomatous infiltration—which may lead to the formation of miliary abscess does not as a rule give rise to symptoms and is rather of pathological than of clinical interest.

Pyelitis is occasionally observed. It may develop in the later stages of the attack or during convalescence. It may involve one or both kidneys. This infection reveals itself by free blood together with

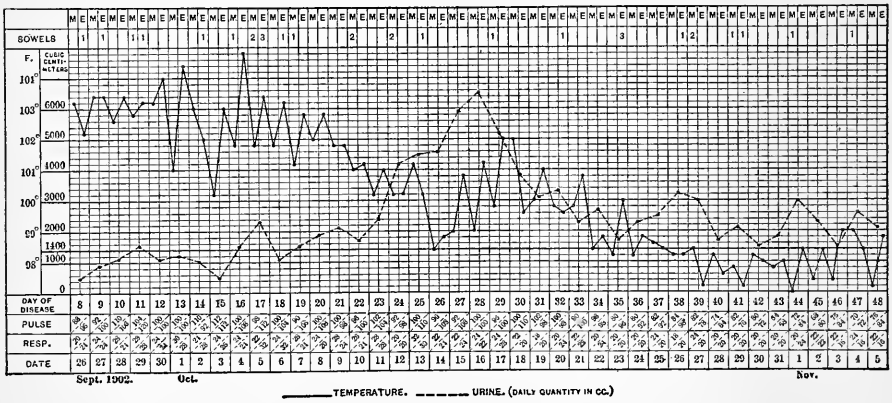


Fig. 211.—Polyuria in enteric fever.

pus and later by a condition in which there is an abundant pyuria. If one kidney only is affected the pyuria may be intermittent, with an almost pus-free urine in the intervals.

Catarrh of the bladder or an acute cystitis may be the cause of the urinary pus. These conditions are somewhat common in enteric fever, especially after repeated catheterization. The more common pyogenic micro-organisms are B. coli, B. typhosus, and staphylococci. Orchitis occasionally occurs, as a rule in association with a purulent urethritis. In women œdema and gangrene of the vulva and acute mastitis are very rare complications. Menstruation is not likely to occur during the attack. When it does occur it is usually profuse.

Hemorrhagic cases of enteric fever have been reported. Of these I have seen several. Hemorrhages into the skin are more common than from mucous surfaces. The general symptoms are severe.

Furunculosis, local gangrene of peripheral parts, periostitis, caries, and necrosis with abscess and sinus formation are complications and sequels often encountered. The bone lesions of enteric fever are notable for their persistence. Typhoid bacilli are present in a majority of the

cases. The tibiæ, ribs, and costal cartilages are most commonly involved. Arthritis involving the knee and hip is among the infrequent complications. It is usually septic.

Typhoid Spine.—Spinal symptoms are sometimes observed in the later course of severe attacks or during convalescence. They consist of pain in the lumbosacral region, aggravated by movement, tenderness upon pressure, stiffness, and inability to execute movements requiring flexion, extension, or rotation of the spine. Nervous symptoms are prominent in some cases. There is no rise of temperature and physical signs are absent. The condition is usually a neurosis—hysterical spine—and terminates in recovery. There may be a spondylitis. I have seen a fatal case of vertebral tuberculosis following enteric fever in a lad of seventeen, which for a period of several months was regarded as an instance of typhoid spine.

Parenchymatous degeneration of the voluntary muscles, which especially affects the recti abdominalis and the adductors of the thigh, sometimes leads to the rupture of the mass of the muscle and may also lead to hemorrhage and abscess formation.

The Association of other Diseases with Enteric Fever.—The frequency of croupous pneumonia as an intercurrent disease has been noted above. Erysipelas occurs in about two per cent. of the cases, more commonly in the period of convalescence. The exanthemata, especially measles and varicella, may be associated with enteric fever.

Malarial fever and enteric fever may coexist. There is no such thing as a hybrid, as indicated by the term typhomalarial fever. Such cases are usually enteric fever or estivo-autumnal fever without well-marked paroxysms.

Tuberculosis as an associated disease has already been mentioned. The various phases of this relation will be fully considered later.

The Effect of Enteric Fever upon Certain Chronic Diseases.—During the attack of enteric fever the paroxysms of epilepsy frequently cease, the irregular movements of chorea and the allied affections are in abeyance, and sugar frequently wholly disappears from the urine in diabetic subjects. In all these conditions the effect is only temporary and the symptoms of the antecedent disease recur with convalescence or shortly afterwards.

Relapse. — The frequency of relapse varies, according to different observers, between three and eighteen or twenty per cent. Relapse occurs with greater frequency in cases treated by systematic cold bathing. It is obvious that a therapeutic method which reduces the mortality will increase the number of convalescent cases in which relapse may occur.

The ordinary form of relapse begins after the defervescence is complete and presents the picture of a repetition of the primary attack, usually shortened and moderated in intensity. The onset is somewhat more rapid; sometimes abrupt with a chill. At times, however, the relapse is even more severe than the original disease and occasionally it terminates in death. The interval between the defervescence and the relapse varies from two or three to twenty days. I have seen a case in which it was five weeks. The question as to whether a repetition of the attack after a prolonged period constitutes a late relapse or an early second attack is purely academic and without practical importance. The relapse is commonly

single; occasionally multiple: two are by no means rare; three are infrequent; five have been observed. The diagnosis of relapse rests upon the range and duration of the fever and its association with rose spots, recurrence of splenic tumor, cessation of the hunger which follows defervescence, and other factors in the enteric fever symptom-complex. It is to be distinguished from recrudescence—a transient fever dependent upon the instability of the heat-regulating apparatus during convalescence and due to trifling causes; from so-called bed-fever,—an unimportant clinical manifestation,—and from the symptomatic fever which may be the first

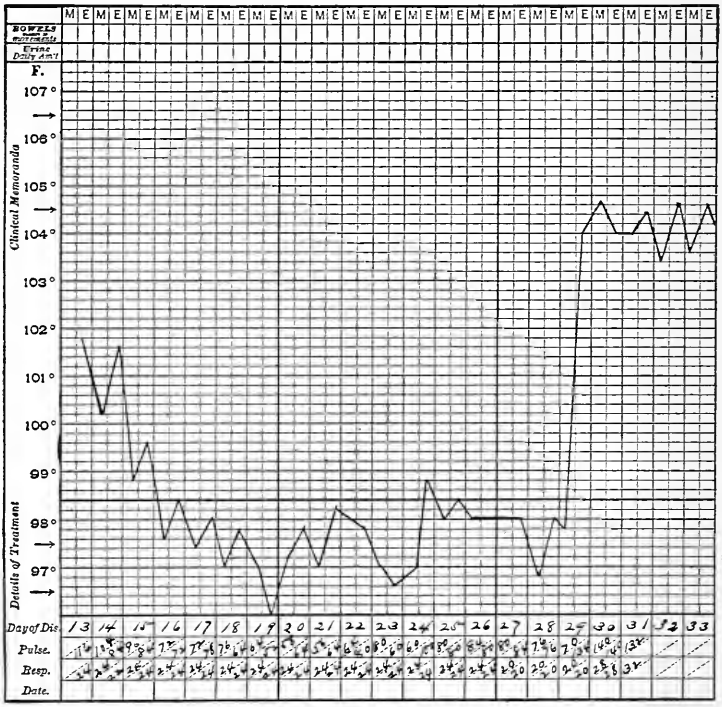


FIG. 212.—Enteric fever. Relapse beginning in abrupt rise of temperature on the 29th day after the onset of the primary attack and the 14th after complete defervescence. Duration of relapse 13 days. Recovery. Girl, aged 11 years.

indication of some sequel or fresh infective or inflammatory process, to which the depraved nutrition of the organism singularly disposes it.

Intercurrent Relapse.—This form of relapse receives its name from the fact that it begins before the primary attack comes to an end. The patient seems to be doing well; the temperature has assumed the remittent, even the intermittent, curve; the tongue begins to clean off; the nervous symptoms ameliorate, and convalescence appears assured, when the fever again rises and becomes subcontinuous, and with recurrence of the symptoms the attack repeats its previous course. Relapses of this form are often severe. They explain a large proportion of the protracted cases.

THEORY OF THE RELAPSE IN ENTERIC FEVER.—In a majority of the cases the circumstances under which relapses occur render it practically

impossible for the reinfection to have come from outside. Neither the water nor the food, including the milk, can be incriminated. The theory of reinfection from within the organism is the only alternative. Of the actual conditions favoring its occurrence little is known. As a working hypothesis, reinfection from the gall-bladder and bile passages as suggested by Chiari appears to meet the requirements of a majority of the cases. The persistence of the bacilli in the mesenteric glands, the spleen, and the gall-bladder, and their presence in the stools for a long time after defervescence favor this view. The immunity conferred by the attack is apparently of slow development. The fact that the greater number of relapses occur within a week or ten days after the fever has subsided, at a time when solid food in increasing amounts is being taken, lends support to this hypothesis. It is at least possible that increased peristalsis may cause the sudden discharge of large quantities of bacilli-laden bile into the intestines at a time when immunity is not yet complete. Too much food and unwholesome food may cause recrudescences of fever, but that they can, in the absence of reinfection, cause relapse is a proposition not to be considered. The hypothesis of Durham, that groups of bacilli of similar species but not identical cause the attack, and that the antitoxin formed in the blood does not neutralize all these groups, those remaining active giving rise to single or multiple relapses, is highly ingenious but lacks proof.

Diagnosis.—(1) The causal or etiological diagnosis; (2) the direct diagnosis; (3) the differential diagnosis; (4) diagnosis by exclusion; and (5) a provisional diagnosis are to be considered.

The diagnosis is a simple matter in well-characterized cases after the first week. The atypical cases are obscure and the more widely they depart from the type the more uncertain does the diagnosis become. The remarkable variations in the disease itself, the great number of complications and sequels, and the irregularity of its course add to the difficulties. The diagnostician must know not only enteric fever but he must also know the many maladies to which in its multiform aspects it bears a close or superficial resemblance.

1. **Causal or Etiological Diagnosis.**—In sporadic cases in which the true nature of the attack remains obscure and the association of cardinal symptoms essential to a clinical diagnosis is lacking, it is of importance to ascertain whether or not the patient has visited a district in which enteric fever is endemic or in which the water supply is tainted; whether he has been in a house in which there were one or several cases or he is a newcomer, and to ascertain the length of time since such probable exposure. The question of acquired immunity is to be considered. A clear history of a previous attack is presumptive but not positive evidence against enteric fever in a doubtful case. In a majority of sporadic cases the source of the infection cannot be traced.

In local epidemics every effort should be made to find the starting-point of the outbreak. Has a case been the source of contamination? Has a sewer burst and discharged its contents into a water reservoir? Do the cases follow the distribution of the milk from a particular dairy? Are they limited to those who have eaten oysters or other shell-fish from beds in sewage-defiled waters? If in a standing camp is the water to be incrimi-

nated, or badly constructed latrines and swarms of house-flies? Upon the answer to questions like these often depend many lives. Diagnosis and prophylaxis, the work of the physician and of the sanitarian, are inseparably bound together. Bacteriologically, the presence of the bacilli in the blood or discharges is of conclusive importance.

The isolation of B. typhosus from the blood by means of culture methods has become in recent years a practical diagnostic procedure. The quantity of blood necessary is considerable but not so great as to do harm in any case. Its withdrawal from a vein is almost painless. This method yields positive results early in the attack, even before the appearance of the eruption. It is especially valuable in septic cases. A recent method is that of Peabody, and consists in the employment of ox-bile as a culture medium for the Bacillus typhosus. The quantity of blood required is small—two cubic centimetres being sufficient. The blood is drawn under antiseptic precautions from the vein at the bend of the elbow and placed in a tube of sterile bile bouillon. This should be sent at once to a laboratory for incubation and bacteriological study. The isolation of a motile bacillus may be regarded as presumptive evidence of the existence of enteric fever, but further cultures are necessary to establish the identity of the organism.

Blood removed from the rose spots contains the bacilli. The procedure is attended with pain and is useless for diagnostic purposes, since the eruption itself constitutes a cardinal diagnostic criterion. The isolation of the bacilli from the urine is now a practical method of diagnosis. Their demonstration is conclusive as regards the nature of the disease. Their presence has been noted in some cases at an earlier period than a positive agglutination test.

The isolation of the bacilli from the stools has been rendered practicable by the more recent culture methods, but the technical difficulties are considerable.

Of the foregoing methods, blood cultures and the examination of the urine are more practical than the others and are in use when the necessary technical skill and the facilities of a clinical laboratory are at hand. The majority of practitioners are not, however, in a position to avail themselves of them.

The Agglutination Test—Widal Test.—This diagnostic procedure is of very great value. It depends upon the property of the blood-serum of an enteric fever patient, when added to a fresh culture of the bacilli, to cause an arrest of the movement of the latter and their agglutination in clumps. The test requires a definite dilution of the serum and time limit. The microscopical examination is made by means of the hanging drop. A dilution of 1–50 and time limit of an hour are in general use. The result is decisive if loss of motility and clumping occur within this period. The dried serum is convenient but its use does not permit of accurate dilution. Small glass bulbs may be used for the collection of the serum. When the test yields a positive result an equally prompt and energetic response to an increased dilution renders the diagnosis even more certain.

The results of the Widal test are to be taken into consideration in connection with the following facts:

A positive result has been obtained some time in the course of the attack or convalescence in about 97 per cent. of cases in which the clinical or post-mortem data rendered the diagnosis certain and in about 93 per cent. of similar cases in which the test was made before the eighth day. In a limited number of cases the diagnosis has been negative in the early course of the attack and become positive in the third or fourth week, or not until after the defervescence or upon the occurrence of a relapse. A well-characterized case of enteric fever with enlarged spleen, rose spots, diarrhœa, even intestinal hemorrhage, may give a negative result throughout. The agglutinating power is retained by the serum for an indefinite time, sometimes months, even years, after the attack. A positive reaction may be therefore misleading, especially in a patient in whom the disease has run its course in an irregular manner or has been of the mildest or of the ambulatory variety—and does not therefore appear in the anamnesis. The blood-serum of healthy persons and of persons suffering from other specific infections, undiluted or in much lower dilution than is used in the Widal test, sometimes causes loss of motility and clumping of the bacilli in cultures, but not with the same promptness and energy, and this property in non-typhoid blood is lost when higher dilutions, 1–50, 1–100, are employed. In cases presenting the clinical picture of enteric fever but in which the Widal test has remained negative, a positive reaction has been obtained with cultures of paratyphoid bacilli — paratyphoid. Caution is therefore necessary, except in the prompt and marked reaction on the one hand and in cases, on the other hand, in which the reaction does not occur at any time, in drawing either positive or negative conclusions. If due regard be paid to the above mentioned peculiarities in the reaction, the Widal test is of value in diagnosis.

This test, important as it is, cannot be made available for the general practitioner because of the difficulty of having constantly on hand fresh cultures of the bacilli. Only in communities in which there are well-equipped clinical laboratories can its full usefulness be realized.

The method of Bass and Watkins consists in the use of formalinized bacilli. The test suspension is composed of 10,000 millions of bacilli per 1 c.c. of 1.7 per cent. NaCl and 1 per cent. formalin. One or two drops of blood diluted with four volumes of water are mixed on a glass slide with an equal quantity of the test suspension. The slide is slowly tilted from side to side. The reaction takes place almost at once. If positive, a grayish sediment is precipitated; if negative, the mixture remains clear.

Macroscopic Method.—This method is based upon the fact that typhoid bacilli are susceptible to agglutination after devitalization by certain antiseptics. The killed bacilli are suspended in narrow test-tubes, forming a homogeneous, slightly cloudy fluid. Upon the addition of typhoid serum agglutination takes place, and flocculi appear which gradually settle as a white precipitate, leaving the supernatant fluid clear. If the reaction is incomplete, flocculi are formed which still float. When the reaction is negative the fluid remains uniformly cloudy, as in the control tube.

Suitable outfits with full directions are made and marketed by the biological laboratories.

The Marris Atropine Test.—The stomach being empty, the pulse-frequency is taken for ten consecutive minutes, while the patient rests quietly in bed. If the number of beats per minute remains practically constant, this is accepted as the average mean rate. Atropine sulphate, grain $^1/_{30}$ or $^1/_{33}$, is then injected hypodermically into the upper arm, after which the patient remains quiet in the same position for twenty minutes. The pulse-rate is then taken again and the counting is continued until the maximum rate per minute has been reached and it has definitely started to fall to a lower level. The difference between this high level and the mean of the ten consecutive minutes before the injection is taken as the release. An increase of only ten beats or less per minute is very suggestive that the patient is suffering from typhoid or paratyphoid infection. A "positive" atropine reaction is shown by little or no increase in pulse-frequency after the injection—ten or fewer beats to the minute. A "negative" reaction is manifest by an increase of fifteen or more. The line between positive and negative is, however, not sharply defined and the test must therefore in doubtful cases be repeated several times. This test is not without value, but there are numerous sources of fallacy.

The Ophthalmic Reaction in the Diagnosis of Enteric Fever.—Chantemesse (1907) announced a new characteristic reaction for the diagnosis of enteric fever. The test is performed by instilling into the conjunctival sac a solution prepared from virulent typhoid bacilli by washing, drying and trituration, precipitation with alcohol, and again drying and reducing to powder. A positive reaction consists in the occurrence, in from three to eighteen hours, of conjunctivitis, which attains its maximum in a few hours and subsides in the course of twenty-four to seventy-two hours. In control cases there is at most a slight hyperæmia which passes off in the course of five or six hours.

The urotoxic coefficient in enteric fever is high and has been found to be increased in cases treated by systematic cold bathing. This fact which is intimately related to the bacteriology of the disease is not used for diagnostic purposes.

2. The Direct Diagnosis.—The presence of B. typhosus in the blood or excretions or a prompt response to the Widal test renders the diagnosis positive independently of the clinical phenomena. The direct diagnosis then rests upon the bacteriological diagnosis. In the vast majority of cases the diagnosis can be made with precision at the bedside—a fortunate matter, since the procedures of the bacteriological laboratory are not always available.

The association of the four following symptoms is of cardinal importance:

(a) The Gradually Ascending Temperature.—This phenomenon is available only when the observations have been begun early in the attack—second or third day. This happens only in a limited number of the cases; but a temperature of 103°–104° F. (39.5°–40° C.) of subcontinuous

type and a history of febrile symptoms of gradually increasing severity and several days' duration are significant. A curve of remittent type in the third week and distinct intermissions with progressively falling evening maxima are of great diagnostic value in a case in which other diagnostic criteria have been uncertain. The duration of the febrile movement is important, but it is in the doubtful cases that we find a shorter or, more commonly, a longer course than in those which are typical.

(b) The pulse-frequency, which is increased, but not in proportion to the temperature. With evening maxima of 104° F. the pulse-frequency may not exceed 90–110 per minute. This derangement of ratio is observed in very few of the acute febrile infections. Yellow fever is conspicuous, but there can scarcely be any question as to the discrimination between that disease and enteric fever. There is nothing characteristic in the pulse itself. Dicrotism occurs under other conditions, but its early appearance is very suggestive.

(c) Enlargement of the Spleen.—This is a constant phenomenon, but cannot always, on account of the distention of the bowel, be demonstrated. It occurs in the other acute infections. Nevertheless enlargement of the spleen at the end of the first week, associated with the other cardinal symptoms, is of great diagnostic value in the direct diagnosis.

(d) The Eruption.—The appearance of the rose spots at the close of the first or in the course of the second week is an event of the highest diagnostic significance. A single spot is without great value, and the single spot is usually a "doubtful" spot; but successive crops of papulomacular, rose-pink spots, distributed over the upper abdominal or lower thoracic regions, disappearing upon pressure or when the skin is made tense, each spot fading in the course of three or four days, do not occur in any other febrile infection and may be regarded as rendering a provisional diagnosis positive.

Symptoms of minor diagnostic importance are epistaxis, the furred tongue, red at the borders and tip, diarrhœa with thin, ochre-colored stools containing now and then one or two hard masses, and separating on standing into a thin upper and a thicker sedimentary layer, tympany, the nervous phenomena, and bronchitis. Intestinal hemorrhage or perforation confirms the diagnosis in a doubtful case. The tendency to complications is characteristic of enteric fever, especially in the later course of the attack. Furunculosis, abscess formation, parotitis, bed-sores, septic phenomena are of merely suggestive importance. A subnormal temperature and eager hunger after defervescence are of very common occurrence. The absence of leucocytosis is of value, but it is precisely in the doubtful cases that inflammatory processes or obscure pus collections impair the value of this method of diagnosis.

Among the symptoms which militate against the diagnosis but do not negative it entirely are marked coryza, herpes, initial sweating, early arthritis and endo- or pericarditis; the continued absence of abdominal symptoms, as diarrhœa, especially if resistant to laxatives, a retracted

abdomen and only slight enlargement of the spleen. The continuing absence of the diazo reaction is not common in enteric fever.

3. **Differential Diagnosis.**—The data for a positive diagnosis of enteric fever are rarely present during the first week. In the absence of a blood culture a provisional diagnosis only is possible. This is especially the case when the patient is seen for the first time. During the fastigium if the four cardinal symptoms of sub-continuous high temperature, slow pulse in proportion to the temperature, enlarged spleen, and rash are present the nature of the malady is beyond question. But it often happens that one or more, even all of these symptoms, are wanting. The fever may be irregular, the pulse rapid, the enlargement of the spleen not demonstrable, and eruption absent. In the later course of the attack septic phenomena, various complications, or an intercurrent relapse may again render the diagnosis obscure.

(a) Diseases which Resemble Enteric Fever in the First Week.

Influenza in some of the cases can be excluded only after several days, especially when the attack has begun with a gradual, step-like elevation of temperature, diarrhœa, and enlargement of the spleen. I have many times seen epistaxis at the onset of an attack of epidemic influenza. The initial nervous symptoms are also much alike. In favor of influenza are a more abrupt onset, catarrhal symptoms, especially coryza and conjunctivitis, the intensity of the headache and its localization in the orbital regions, a pulse-rate proportionate to the rise in temperature, and the fact that the attack runs its course and defervescence is complete by the end of a week.

Febricula.—Every practitioner sees cases of transient fever with headache, malaise, anorexia, and sometimes barely recognizable enlargement of the spleen. If the symptoms last twenty-four hours and disappear altogether, the attack is known as ephemeral fever; if they continue longer, to six or seven days in the absence of local trouble, it is designated febricula. Many of the cases are examples of the mildest variety of enteric fever. In some instances rose spots are seen. The illness may be due to some other infectious agent, or gastro-intestinal catarrh, or symptomatic of some obscure local infection or inflammatory process.

Acute Exanthemata.—Scarlet fever, measles, and the variolous diseases may during the period of onset give rise to the suspicion that enteric fever is developing. The character of the temperature range, the coryza in measles, the angina of scarlatina, the intense headache and backache of variola, together with the initial rashes when present and the appearance of the definite eruption in a relatively short time, settle any question as to the essential nature of the infection.

Febrile Enteritis and Gastro-enteritis.—As a rule, gastric and intestinal catarrhs run their course without fever. Febrile cases do, however, occur. It is to the infrequent cases of this kind that such terms as gastric fever and mucous fever owe their existence. Most of the cases so designated by practitioners are cases of enteric fever, and these terms are, fortunately, falling into disuse.

Appendicitis.—The gastro-intestinal symptoms and especially the pain and tenderness may if attended by a rise in temperature simulate enteric fever. I have known several instances in which a patient suffering from the latter disease has been admitted to a hospital at night and at once operated upon, with the recognition upon the following day of the true nature of the disease. The sudden onset, the localization of the pain, and tenderness, the absence of fever, or its irregularity when present, and the lack of the cardinal symptoms upon which the diagnosis of enteric fever rests should put the practitioner upon his guard.

Right tubo-ovarian disease with fever may also simulate enteric fever. The presence of a tender mass upon the right side with fixation of the uterus and leucocytosis are of positive diagnostic significance.

Meningotyphoid — cerebrospinal fever — takes first place among the diseases which simulate enteric fever in its more irregular forms. In fact the resemblance between the cerebrospinal form of enteric fever in the first week and cerebrospinal fever is so great that a differential diagnosis can only be made by laboratory methods—blood culture. The onset is sudden with intense headache, photophobia, delirium, painful rigidity of the back of the neck, and sometimes vomiting. Kernig's sign may be present. Examination of the fluid obtained by lumbar puncture may show the meningococcus. The appearance of rose spots and abdominal symptoms at the end of the first week and mitigation of the nervous symptoms are characteristic of enteric fever. Herpes is common in cerebrospinal fever.

Pneumonia.—Pneumotyphus is very rare. The sudden onset with chill, high fever, pain in the side, cough, and the signs of consolidation are very misleading. The later course is that of enteric fever. The difficulties are increased in the irregular cases of enteric fever in aged persons. Such cases have been regarded as pneumonia until at the autopsy the intestinal lesions of enteric fever have been found. The recognition of intercurrent croupous pneumonia in the course of the disease is a comparatively easy matter provided that systematic routine examinations by the methods of physical diagnosis are made.

There are cases of central pneumonia, occurring independently of enteric fever, which simulate it very closely. These cases run their course for days with no other symptoms than those of fever and perhaps a trifling cough. Pain, rusty sputum, and the signs of consolidation are not present. The diagnosis rests upon the abrupt onset with chill and high temperature, difficulty in breathing, the early appearance of herpes, and a slight degree of jaundice. Leucocytosis is of diagnostic value. It is to be remembered that this sign may be absent in the gravest cases of pneumonia and present in enteric fever complicated by inflammatory or purulent processes. The absence of the temperature curve of enteric fever, of relative slowness of the pulse, of considerable enlargement of the spleen, and of rose spots is important.

Nephrotyphus.—The cases which begin with the clinical phenomena of an acute nephritis present great diagnostic difficulties during the first week.

There are headache, vertigo, mental dulness, disinclination for effort, loss of appetite, and fever. Epistaxis occurs in both conditions. The illness looks like enteric fever; the urine is that of an acute inflammation of the kidneys. It is scanty, high-colored, of high specific gravity—1.024 to 1.030—and contains much albumin, together with hyaloangular and epithelial tube-casts, cylindroids, and red blood-corpuscles. Rose spots, splenic tumor, a more or less characteristic temperature range, and the duration of the attack render the diagnosis clear. The nephritis does not tend to become chronic.

Sepsis.—This term includes septicæmia, pyæmia, and septicopyæmia. In general the differential diagnosis between these conditions and enteric fever is not attended with difficulty. Where there is trauma or obvious bone disease or demonstrable suppuration no question arises. There are, however, forms of sepsis, especially those of cryptogenetic origin, having in common with enteric fever high temperature, splenic tumor, and nervous symptoms in which the diagnosis is very uncertain. Among the symptoms which favor the diagnosis of sepsis are the following: irregular fever with marked remissions and intermissions early in the illness; chills followed by profuse perspiration; endocarditis of the septic or malignant form; septic arthritis involving a single joint or many—tenderness upon pressure over the bones—sternum, clavicles, tibiæ—and retinal hemorrhage. The pulse also is very frequent and arrhythmic. In many of the cases there are marked meningeal symptoms and cutaneous lesions, herpes, urticaria, erythema, and petechiæ are common. Sepsis frequently occurs in the course of enteric fever, and there is a recognized form of typhoid septicæmia—facts which are of importance in the diagnosis of individual cases.

(b) Diseases which Resemble Enteric Fever in the Fastigium.—*Malarial fever* may as a rule be readily differentiated from enteric fever. The regularly intermitting forms present no difficulties. In the estivo-autumnal form the diagnosis may be uncertain for several days. The appearance after a time of the parasite in the blood settles all doubt as to the nature of the disease. Meanwhile the absence of chills, the continued fever with very moderate remissions, together with weakness, diarrhœa, and a palpable spleen, suggest enteric fever. The malarial and the typhoid infection may be present in the same patient at the same time. With the estivo-autumnal variety this association is not uncommon, as was shown in soldiers returning from Cuba and Porto Rico during the Spanish-American War. With the tertian and quartan parasites the association is rare and these organisms are very seldom present in the blood of individuals suffering from enteric fever. A hybrid disease such as is indicated by the term typhomalarial fever—a separate nosological entity—does not exist.

Typhus fever may be differentiated from enteric fever by the eruption, which in the latter is far and away more sparse and appears several days later in the course of the attack. Cases in which the rash appears early and is so copious as to suggest typhus fever do occur, but they are extremely rare. It does not, save in the rarest cases of hemorrhagic enteric fever, become petechial, as is the rule in typhus. The abrupt rise and

critical fall of temperature in typhus are very significant, as is also the high pulse-frequency. Dicrotism is often present in both diseases. The difference in the duration of the two diseases, typhus lasting usually from ten to fourteen days, is to be noted. The Widal reaction is almost invariably wholly absent in typhus. Blood cultures may become necessary in a doubtful case.

Relapsing fever may be readily differentiated from enteric fever by its abrupt onset with chill and very high temperature, jaundice, pain and tenderness in the epigastric zone, critical defervescence, period of complete apyrexia, and relapse. The presence of the spirochæta of Obermeier in the blood is absolutely conclusive. In typhus and relapsing fevers the prevalence of an epidemic is to be taken into consideration in the differential diagnosis.

Internal anthrax presents the symptoms of a severe infectious disease with intestinal symptoms. Fever, diarrhœa, and splenic enlargement occur. There are symptoms, however, which scarcely belong to enteric fever, as repeated vomiting, colic, bloody diarrhœa, hæmaturia, dyspnœa, cyanosis, and submucous extravasations of blood in the mouth. An examination of the blood reveals the presence of anthrax bacilli.

Acute miliary tuberculosis is occasionally mistaken for enteric fever. This error in diagnosis arises from the fact that the former disease frequently begins rapidly in persons apparently in good health, with fever, enlargement of the spleen, and nervous symptoms, and without demonstrable signs of organic lesions upon physical examination. The presence in rare cases of a scanty eruption of rose-colored maculopapules not to be distinguished from the rash of enteric fever adds greatly to the uncertainties of the diagnosis. Their recurrence in crops is in favor of the latter affection. The Widal test should be tried and, in the case of a negative result, repeated at intervals of some days. In such cases there is usually little sputum and neither that which is expectorated nor the urine contains tubercle bacilli. Careful examination of the chest will often elicit suggestive signs, as vesiculo-tympanitic resonance at an apex and a few scattered small mucous or coarse crepitant râles of high pitch. Cyanosis and dyspnœa are prominent symptoms. The pulse, in the absence of an associated meningitis in which it is often slow, is frequent, feeble, and arrhythmic, showing in particular remarkable variations in frequency in the course of brief intervals of time. The splenic enlargement is less marked than in enteric fever; but there are a few cases of the latter disease in which the spleen is but little enlarged and many in which during the fastigium the enlargement cannot be demonstrated on account of the meteorism. A complicating meningitis may occur in either affection, but is much more common in tuberculosis. If an ophthalmoscopic examination, which should be repeated from time to time, reveals the presence of tubercles in the choroid, the diagnosis is established. The atypical course of the temperature in tuberculosis, especially its extreme irregularity, the occurrence of remissions, and its occasional morning exacerbations and evening remissions—inverse type—are of great diagnostic value.

Tuberculous peritonitis may, in certain of its forms, present a misleading resemblance to enteric fever. The attack begins gradually with abdominal tenderness, meteorism, and diarrhœa. There are moderate fever of subcontinuous or remittent type and rapid wasting. The diagnostic criteria are those already mentioned under acute miliary tuberculosis. Ascites, a doughy distention of the abdomen, the presence of enlarged mesenteric glands or a sausage-shaped omental tumor are confirmatory data in tuberculous disease.

Sepsis may present the same difficulties in diagnosis from enteric fever in this period as in the first week. The points of differentiation are the same and have already been set forth in sufficient detail. The long-continued symptomatic fever of deep-seated suppuration, often obscure, may in the absence of chills and sweating closely simulate enteric fever. This is especially true of the deep abscesses which occasionally occur in visceral and bone tuberculosis.

Malignant endocarditis is not rarely mistaken for enteric fever. Changing murmurs, embolism, and the presence of leucocytosis are of great diagnostic aid. The Widal reaction and blood cultures when positive dispel any doubts as to the presence of enteric fever.

(c) Diseases which Resemble Enteric Fever in its Later Course. —Septic conditions, various complications, and intercurrent relapse greatly modify the period of decline. In fact the terminal course of an ordinarily well-characterized, uncomplicated attack of enteric fever is often as typical as the onset. The falling temperature with its remittent and intermittent curve, the cleaning tongue and urgent hunger, the clearing mind and natural sleep, all coming on toward the close of an illness of three or four weeks' duration, would almost justify a diagnosis in the absence of a history of the previous course of the attack.

Sepsis of obscure origin—cryptogenetic—sometimes *sepsis due to obvious causes,* as purulent effusion, abscess formation, or caries, may so dominate the clinical picture as to raise a doubt in regard to the true nature of the primary attack. In default of a satisfactory anamnesis the methods of the laboratory, especially blood cultures, the examination of the urine for B. typhosus, and the Widal test are in many cases essential to a positive diagnosis. Complications, as pleurisy with effusion, bronchopneumonia, pulmonary abscess or gangrene, malignant endocarditis, cystitis and pyelitis, and various nervous diseases may assume such a degree of prominence as to dwarf the significance of the early symptom-complex and raise the question as to whether or not the previous symptoms have been those of enteric fever or simply earlier manifestations of the present disease.

Intercurrent relapse frequently prolongs the attack to six or seven weeks. Fresh crops of rose spots, the character of the temperature range, which may after having been strongly remittent again become subcontinuous, the persistent enlargement of the spleen, and the other symptoms of a specific rather than a septic infection afford the criteria for a diagnosis.

Uræmia in its chronic forms may suggest enteric fever at the later periods of its course by a rapid and feeble pulse, dry and fissured tongue, stupor, wandering delirium, subsultus, and continued fever of mild type.

The urinary findings, the condition of the arteries, a negative Widal reaction, and the previous history are usually sufficient for the differential diagnosis.

4. **Diagnosis by Exclusion.**—Enteric fever is by far the most common of the febrile infectious diseases; with the exception of intestinal symptoms, enlargement of the spleen, trifling bronchitis, and a relatively slow pulse, there are no constant evidences of visceral disease; the age at which the disease is most common and the immunity which is established by the attack are facts available in a doubtful case for the diagnosis by exclusion.

5. **A Provisional Diagnosis.**—There are cases in which a positive diagnosis cannot be made when the patient is seen for the first time. Delay may be required for the accumulation of the necessary data in the progress of the attack to a point at which characteristic symptoms appear. A provisional diagnosis becomes, under these circumstances, imperative. Pending the decision a due regard of the welfare of the patient and the community demands the exercise of all the measures of treatment and all the precautions against the spread of the disease that we would employ if a positive diagnosis were made.

The Diagnosis of Intestinal Perforation.—The direct diagnosis of this accident rests upon the association of the symptoms to which it gives rise. There are cases, however, in which several of the more characteristic symptoms are not present, and every clinical phenomenon of intestinal perforation may show itself in the course of enteric fever in perforative lesions of other organs, as the appendix, gall-bladder, or peptic ulcer of the stomach or duodenum; while acute abdominal symptoms with or without collapse, followed by the signs of local or general peritonitis, may be the manifestations of intussusception, volvulus, strangulation of a Meckel's diverticulum, softened splenic infarct, hepatic abscess or pseudo-abscess of the mesenteric glands. Prompt recourse to surgical measures in any of these conditions may be the only means of saving life. In the face of the urgent symptoms of some grave intra-abdominal accident it is better in selected cases not to lose time in the attempt to make a diagnosis of the lesion by ordinary measures but at once to open the abdomen and ascertain the actual condition and if possible correct it. This diagnostic procedure is justified by the fact that enteric fever patients bear anæsthesia and operation well.

Prognosis.—The mortality varies greatly in different outbreaks, the range being from five to seventeen per cent. It is slightly higher in hospital than in private practice. After the first year, the prognosis is in general less favorable as the age of the individual increases. The mortality is greater after puberty; after forty it rises rapidly and in aged persons enteric fever is a very fatal disease. In respect of prognosis the infantile type—high evening temperatures with marked morning remissions throughout—is much more favorable than the adult type of the disease—high or moderate evening exacerbations with very slight morning remissions. Fat persons do not bear enteric fever well. Their powers of resistance to infections in general are less than in the spare and muscular; there is an increased tendency to parenchymatous degenerations of the viscera; the evidences of myocardial changes are early noted, and the nursing and treatment are less satisfactory. Those given to the abuse of alcohol also

bear the disease badly. Women in general show a higher mortality than men, and pregnant and lying-in women attacked by enteric fever are in great danger. When the disease develops in persons suffering from chronic disease, especially affections of the heart, chronic bronchitis, emphysema, goitre, and pulmonary tuberculosis, the prognosis is less favorable than in those in previous good health. When the tuberculous patient survives the attack, the lung trouble usually manifests itself with increased intensity. There are, however, exceptions to this rule. I have seen several cases in which an apparent arrest took place after convalescence. An antecedent chronic nephritis adds to the gravity of the case. Diabetes mellitus is also unfavorable. The temperature is not usually high and sugar disappears from the urine, but the resisting powers of the patient are much impaired.

The intensity of the infection as shown by high temperature maxima, the limited range of the remissions, prolongation of the fever, and the prominence of nutritional disorders and nervous symptoms is of prognostic importance. The outlook is relatively much more favorable in the cases in which the fever is moderate and the associated symptoms of mild degree—typhus levissimus—and in those in which, while the fever is high and the morbid phenomena severe, the course is short—abortive cases. In general an abrupt rise of temperature occurs in the latter group of cases and is therefore not without value as indicating a short attack. Even in these cases a severe complication, a relapse, or the gravest accidents—hemorrhage, perforation—may occur. These events are, however, much more common in cases otherwise severe.

The maintenance of the power of the heart as indicated by the pulse is very important in prognosis. So long as the frequency remains low as compared with the fever and the volume is fair, the outlook is relatively favorable; but an increased pulse-frequency, associated as it almost invariably is in this disease with loss of power, is of grave prognostic significance. This is particularly the case when there are developed at the same time such evidences of circulatory failure as pulmonary hypostasis, faint cyanosis, coolness of the extremities, pulmonary œdema, and symptoms of collapse. The prognosis becomes progressively more ominous as the frequency of the pulse increases. In children and neurotic individuals, especially women, a frequent pulse is less significant, particularly when it again becomes slower.

As a general rule the gravity of the case is proportionate to the intensity of the nervous symptoms. Continuing delirium, stupor, coma, and particularly coma vigil, are of grave import; so also are meningeal symptoms, apoplectiform seizures, and local and general convulsions. Less alarming are the psychic derangements which occur toward the close of the attack and during convalescence—postfebrile insanity.

The prognosis is rendered unfavorable by the development of complications and sequels. These are, as has been already stated, more numerous than in any other infectious febrile disease and not infrequently determine the outcome of the case.

Intestinal hemorrhage, when slight and occurring early in the attack, is not necessarily unfavorable. Large bleedings occurring early are distinctly so, for four reasons: they indicate grave local lesions and usually

also intense infection; they debilitate the patient and lower his powers of resistance; they are often repeated after a longer or shorter interval and in some instances are followed by perforation, and finally they constitute a distinct contraindication to the treatment by systematic cold bathing.

Intestinal perforation is almost always followed by the fatal issue. The patient succumbs in the course of a few days to the consecutive peritonitis. In rare instances a longer period may elapse and a very few cases of spontaneous recovery have been noted. A favorable prognosis cannot be made and the only chance for the patient lies in prompt surgical intervention. Peritonitis without perforation constitutes a complication of most gloomy import. It is probable that a proportion of the cases described as perforation with recovery have been instances of this kind.

Finally the prognosis is much influenced by the general management of the individual case and treatment. Skilful nursing, a careful dietary, the avoidance of drugging, and systematic cold bathing have reduced the death-rate to about seven and one-half per cent. The earlier the treatment is instituted the better the result.

PARATYPHOID FEVERS.

Definition.—A group of infectious febrile diseases, caused by organisms intermediate between Bacillus typhosus and Bacillus coli and presenting the clinical phenomena of enteric fever.

Etiology. — There are a number of organisms in this intermediate series, including the Bacillus enteritidis, and several varieties causing diseases in animals. Buxton has suggested the following classification: "PARACOLONS.—Those which do not cause typhoidal symptoms in man. A group containing many different members but culturally alike. PARATYPHOIDS.—Those which cause typhoidal symptoms. (A) A distinct species culturally unlike the paracolons. (B) A distinct species culturally resembling the paracolons."

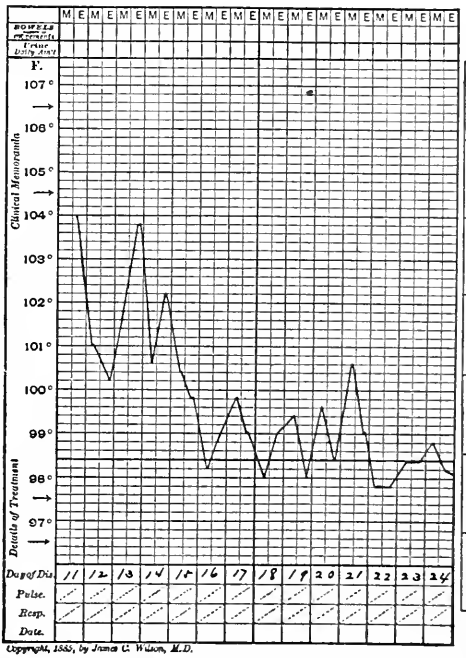


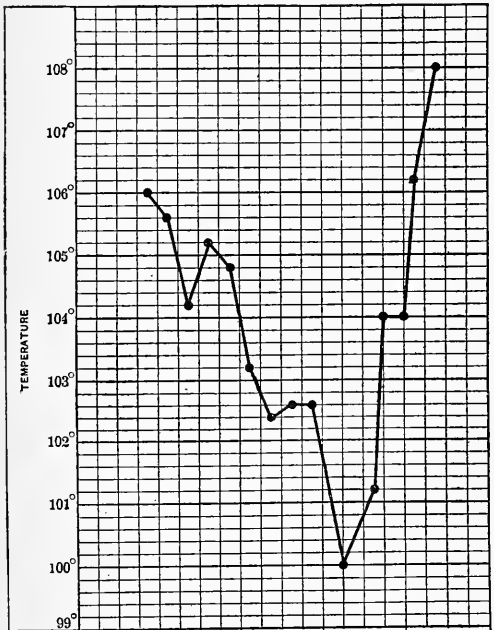
FIG. 213.—Paratyphoid fever.—Jefferson Hospital.

Cases of paratyphoid have been reported from all parts of the world in which systematic laboratory work in bacteriology is carried on. It has occurred in series of enteric fever cases, in house epidemics, and under circumstances which render it probable that it is sometimes a water-borne disease.

Symptoms.—The features of reported cases are variable.

1. Many of the cases cannot be distinguished from enteric fever except by the failure of the blood-serum to agglutinate B. typhosus and its power to agglutinate the organisms of this group. Hemorrhage, crural phlebitis, and relapse occur. 2. Others present the clinical features of septic infections and resemble the so-called typhoid septicæmia or enteric fever with intercurrent or terminal sepsis. The diagnosis is reached by exclusion rather than by the ordinary clinical features of enteric fever. 3. Finally the organisms have been found in abscesses in cases in which no history of enteric fever has been obtained.

The first group of cases, those which are clinically indistinguishable from enteric fever, are almost always mild and terminate in recovery. The anatomical lesions are therefore as yet undescribed. The last group is without interest in this respect. Cases of the second group may end fatally. Wells and Scott (1904) studied a fatal case of their own in connection with four fatal cases collected from the literature, one of which occurred in my service in the Pennsylvania Hospital and was studied by Longcope. The most constant lesion was enlargement of the spleen. The intestinal conditions were variable. In two of the cases the intestines were normal. Ulcers were present in the others, but they resembled those of dysentery rather than of enteric fever. In all the cases the solitary follicles, the Peyer's patches, and the mesenteric glands were unaffected. The other changes present were those of a septicæmia.



DAY OF DIS.	8		9		10		11		12	
PULSE		128	120	132	124	130	124	120	164	
RESP.		36	32	32	36	30	36	36	48	
DATE	March 9		10		11		12		13	

FIG. 214.—Fatal paracolon infection.—Pennsylvania Hospital.

Diagnosis.—The direct diagnosis of paratyphoid rests upon the failure of the blood-serum to agglutinate B. typhosus and its power to agglutinate B. paratyphosus or B. paracoli. It is in the highest degree probable that reported cases of mild enteric fever and cases of the so-called septicæmic variety have been instances of paratyphoid. That conditions not to be clinically differentiated from enteric fever may be caused by a number of allied pathogenic organisms has been established.

II. TYPHUS FEVER.

Typhus Exanthematicus; Jail, Camp, or Ship Fever.

Definition.—An acute, infectious, epidemic disease characterized by sudden onset, intense fever, a peculiar rash, at first macular, later petechial, great prostration, marked nervous symptoms, a defervescence usually critical, and an average duration of fourteen days. There are no characteristic anatomical lesions.

Typhus—τῦφος smoke—used by Hippocrates to define a confused state of the mind with a tendency to stupor, expresses a prominent condition of the disease. Typhus is now a rare disease. Its practical disappearance dates from the period when enteric fever became prevalent. Sporadic cases and small local epidemics of typhus occasionally occur in great cities, particularly in Great Britain and Ireland and in Russia.

Etiology.—Predisposing Influences.—Typhus is a disease of cold and temperate climates. Its prevalence is but little influenced by the season of the year or by meteorological conditions. Overcrowding, domestic and personal filthiness, insufficient food, and intemperance are important predisposing influences to typhus. It is a disease of the poor and under-fed of large cities. Age and sex are without influence. Occupation is without influence except as it involves actual exposure to the contagion, as in the case of hospital attendants, physicians, clergymen, and those who care for the dead.

The Exciting Cause.—The infecting agent is Bacillus typhi-exanthematici. "This organism is a small, pleomorphic gram positive bacillus, not motile, not encapsulated and not acid-fast. Its length varies from .9 to 1.93μ, its breadth being from one-fifth to three-fifths its length." It does not produce spores and is an obligatory anaërobe. It is present in the blood during the fever and can be transmitted by inoculation to monkeys and guinea-pigs. The disease is usually communicated by the bite of the body-louse (*Pediculus vestimenti*) or the head-louse (*P. capitis*). This fact, first discovered by Ricketts, who died of typhus contracted during his investigations, and Wilder, fully explains the distribution and prevalence of the disease and points to a direct and efficient prophylaxis.

Symptoms.—The period of incubation is about twelve days. It may be less. Prodromes occasionally occur.

Stage of Invasion.—The onset is abrupt and marked by a chill or chilliness, followed by fever. The face is flushed, the eyes injected, headache is constant and severe. The patient complains of great pain in the back and soreness of the limbs and joints. Early muscular weakness, an extreme sense of prostration, confusion of mind and failure of memory occur. Delirium is an early symptom; it may be mild and wandering or active and noisy. The tongue, at first large, pale, and coated with a thick fur, presently becomes brown and dry. A stale, unpleasant odor loads the breath. Constipation is the rule. The pulse is full but compressible. It soon grows feeble and varies in frequency from 120–130. Dicrotism is uncommon. The temperature rises rapidly. By the third or fourth day it may reach 103°–105° F. (39.5°–40.5° C.), and continues at this range with moderate morning remissions until the crisis. Hyperpyrexia, 107°–109° F. (42°–42.7° C.), not infrequently precedes death.

Stage of Eruption.—On the fourth or fifth day, rarely later, the eruption appears. It consists of numerous roseola-like spots of irregular outline and from one to three lines across, scattered singly or arranged in close-set groups like the rash of measles. These spots are at first of a dirty rose color, slightly raised above the surface of the surrounding skin, and disappear upon pressure. In the course of a day or two they become darker and are then no longer elevated but appear as faint, dirty brown stains without defined margins. A little later petechiæ show themselves at the centre of many of these spots. The spots fade during the first half of the second week and disappear with or without desquamation toward its close. In many of the cases petechiæ appear about the time the typical rash begins to fade. A faintly reddish, lightly defined mottling or marbling of the skin between the spots or groups of spots also occurs. This mottling has been described from its appearing to lie beneath the surface as the subcuticular eruption of typhus. The appearance of the rash varies greatly, the differences being determined by the general abundance of the two eruptions, by the relative preponderance of one or the other, and by the extent of the petechiæ. The spots and mottling together constitute the "mulberry rash" of Jenner.

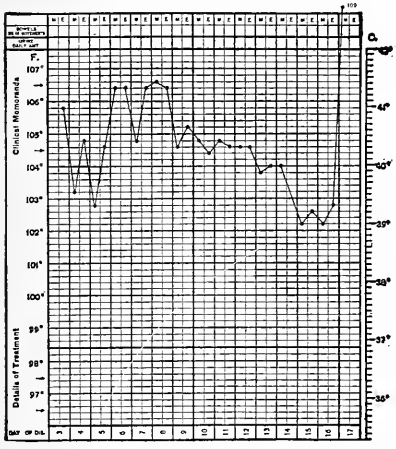


Fig. 215.—Typhus fever—fatal on 16th day of attack.

The eruption usually appears first on the sides of the chest or abdomen and spreads in a brief time over the body and limbs. It rarely occurs upon the neck or face. It may first appear upon the back of the hands. The roseola-like rash may be absent altogether, the faint subcuticular mottling alone being present. The entire absence of eruption is very rare. Sudamina are not common. The odor surrounding the patient has been regarded as characteristic.

Stage of Nervous Prostration.—During the second week the depression becomes profound. Headache passes into delirium; drowsiness and stupor are marked and in severe cases there is a tendency to coma. The delirium continues. Coma vigil, subsultus tendinum, and picking at the bedclothes may occur. The conjunctivæ are deeply injected; the pupils contracted; deafness is common. The flushing of the face gives place to a dusky pallor and emaciation progresses. There is cough and râles are heard in all parts of the chest. Hypostatic congestion occurs. The heart's action is faint; the pulse small, weak, often difficult to count—ranging from 112–140 or more. A systolic murmur in the mitral area is not infrequent. The area of splenic dulness is increased. The state of the bowels now varies from constipation to irregular, scanty dejections or moderate diarrhœa. The urine is decreased in amount, high colored, and frequently albuminous. In severe cases the discharges are passed involuntarily or there is retention of urine. Areas of the skin subjected to pressure show a tendency to slough. The surface now becomes cooler and is often moist.

Death in the malignant cases occurs in the course of a few days—*typhus siderans;* more commonly between the tenth and seventeenth days. The mode of death is by coma or by asphyxia in consequence of sudden pulmonary engorgement or by failure of the heart, the pulse becoming imperceptible, the surface cold, livid, and bathed in sweat. In the milder cases and especially in children the rash is slight, petechiæ are absent, and defervescence takes place at the end of the first or the beginning of the second week. In the average cases defervescence takes place about the fourteenth day by crisis, the temperature falling in a single night or in the course of twenty-four or forty-eight hours to the normal or even below it. Convalescence is rapid, relapse extremely rare.

Complications and Sequels.—Laryngitis, bronchitis, and bronchopneumonia are common; gangrene of the lung may occur. Independently of scurvy, which has been a frequent concomitant in typhus epidemics, bleeding from the nose, gums, bowels, urinary passages, and the vagina has been noted, as well as the spitting and vomiting of blood. In certain epidemics gangrene of the extremities, the nose, and the genitalia, and cancrum oris have occurred. Septic parotitis and arthritis and subcutaneous abscesses occur. Various palsies are met with.

Diagnosis.—The DIRECT DIAGNOSIS of typhus during epidemics is usually a simple matter. In early or isolated cases the nature of the disease must remain in doubt until the appearance of the eruption. The abrupt onset, initial chill, and sudden rise of temperature are important. The critical defervescence about the fourteenth day is characteristic.

DIFFERENTIAL DIAGNOSIS.—ENTERIC FEVER (see p. 30). RELAPSING FEVER has often prevailed in connection with typhus. The stage of complete apyrexia, the clear mind, epigastric pain and tenderness, absence of eruption, low death-rate, and the spirochætæ of Obermeier serve to differentiate this disease from typhus. CEREBROSPINAL FEVER may at the onset resemble typhus. Associated headache, vomiting, and painful rigidity of the muscles of the back of the neck, Kernig's sign, and in fatal cases characteristic lesions are of diagnostic importance. The presence of the Diplococcus intracellularis meningitidis in the fluid withdrawn by lumbar puncture will determine the question. PLAGUE.—Nausea and vomiting, pallor, and the early appearance of glandular swellings are characteristic. The duration of the plague is much shorter than that of typhus and the mortality greater. MALARIA.—The malignant malarial fevers of tropical and subtropical climates occasionally present strong resemblances to typhus but they are endemic, not contagious, unattended by specific eruptions, show greater enlargement of the spleen and in the blood the malarial parasite. MEASLES and typhus in children are attended by a somewhat similar eruption about the fourth day of the attack. In measles catarrhal phenomena are prominent during the stage of invasion; the eruption, which first shows itself upon the face, is brighter in its tints and rarely petechial. ALCOHOLISM.—Certain cases are attended by trembling delirium like that occasionally seen in typhus. Shivering, headache, pains in the limbs, fever, and eruption are absent.

Prognosis and Mortality.—The mortality ranges from 10 to 20 per cent. It is much influenced by age; not exceeding 4 per cent. under ten

years and rising above 50 per cent. after sixty. Individual peculiarities unfavorably affecting the prognosis are intemperate habits, disease of the kidneys, gout, obesity, and mental depression.

TABARDILLO: *Mexican Typhus.*—This infectious disease is identical with European typhus. The period of incubation appears to be longer; the onset is less abrupt and the defervescence more commonly by lysis than by crisis. The eruption is less often petechial. The duration of the febrile movement is about the same and the intense headache and backache, prostration, vertigo, gastro-intestinal symptoms, apathy and delirium correspond to the typhus of Europe. McCampbell, who has carefully studied the subject, believes tabardillo to be a variety of typhus, the departure from type being due to the influence of temperature and other climatic conditions. This observer found a marked leukocytosis in all the cases examined, exceeding 30,000, in three cases terminating fatally. The erythrocytes were normal or slightly increased; the hæmoglobin ranged from 85 to 95 per cent. and the coagulativity of the blood was much decreased. The post-mortem findings are not characteristic.

BRILL'S DISEASE.—Brill (1898) reported a series of cases of acute infection closely resembling enteric fever but lacking the characteristic phenomena of that disease. He subsequently extended the series to 221 cases and gave a detailed description of it, highly suggestive of typhus. Lewis has reported cases observed in Philadelphia, where I have also seen a number. The cases are usually admitted as enteric fever. They are characterized by intense headache, apathy and prostration, an abundant erythematous, papular eruption and a subcontinuous pyrexia terminating in crisis or rapid lysis in two weeks or less. In rare instances two or more cases have occurred in the same dwelling. It has not occurred immediately or remotely in other persons when cases have been treated in the general wards. The case mortality is low. The identity of the disease described by Brill with typhus fever has been definitely determined. (Anderson, Goldberger.) Its sporadic occurrence in the cities of the Atlantic seaboard of North America is due to immigration from Southeastern Europe; its failure to prevail in epidemics to the cleanlier habits and diminished lousiness among the Americanizing people.

III. RELAPSING FEVER.

Febris Recurrens.

Definition.—An acute, infectious, epidemic disease caused by the spirochæta of Obermeier, characterized by a febrile paroxysm of five to seven days terminating by crisis, an interval of complete apyrexia of about the same duration and one or more abrupt relapses. There are no characteristic anatomical lesions.

Relapsing fever has prevailed extensively in Europe and particularly in Ireland, usually in association with typhus fever. It has occurred in India and other tropical countries. Brief outbreaks have occurred in America. The clinical manifestations are the same, but the blood parasites present different characteristics. The variety in the European relapsing fever is *S. obermeieri;* in the African *S. duttoni;* in the American *S. novyi;* in the Indian, *S. carteri.* An endemic form of relapsing fever has been recently found to exist in Colorado. (1918.)

Etiology.—Predisposing Influences.—Destitution, filth and overcrowding play an important part. Climate and season have no direct influence upon the development or propagation of relapsing fever.

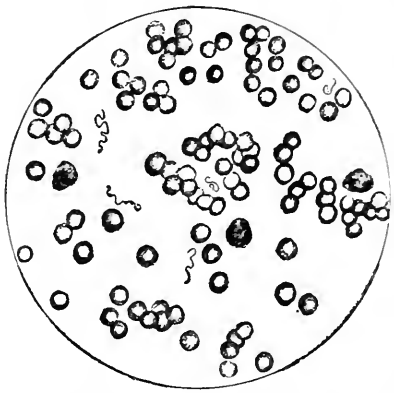


Fig. 216.—Spirillum of relapsing fever in blood.

The Exciting Cause.—Obermeier in 1873 demonstrated in the blood an organism which is the specific cause of relapsing fever. This microorganism is a slender spirillum or spirochæta varying in length from 16 to 40μ, twisted spirally in from ten to twenty turns. In fresh blood it is very active. Under a low power it shows itself by the commotion among the blood-corpuscles, caused by its rapid movements. The spirochætæ are present in the blood only during the febrile paroxysm. About the time of the crisis they disappear and are not found during the apyrexia. At this period minute, highly refractive bodies stated to be spores are seen in the blood. Upon the occurrence of relapse active spirochætæ are again found in the blood. Relapsing fever may be produced in man by inoculation with the blood of a patient, and several instances are recorded where infection has followed wounding of the hands at autopsies. Koch, Van Dyke Carter, and others have produced the disease in monkeys by inoculation. Among suctorial insects lice alone can transmit European relapsing fever. "Bedbugs are never known to transmit the infection by their bites." (Noguchi, 1917.) Contact infection does not occur. In monkeys killed ten hours after the crisis the parasites are found in the phagocytes in the spleen. They are not present in the secretions or excretions. They have been found in the blood of the fœtus. Second and third attacks in the same individual in the course of a few months have been observed.

Symptoms.—Incubation varies from five to seven days. It is exceptionally only twenty-four hours. The Primary Paroxysm.—Prodromes are as a rule absent. The onset is marked by chills or chilliness, rapid rise of temperature, headache, and pain in the back and limbs. Sweating is common. Appetite is lost and nausea and vomiting are sometimes persistent. The tongue is usually moist, covered with a thick white or yellowish-white fur. It is apt to continue in this condition throughout the paroxysm. Rarely it becomes dry or shows a dry brownish streak in the middle. The bowels are constipated. In many cases jaundice occurs. There is no characteristic eruption. Sudamina and facial herpes occur. As early as the second day there is distress in the epigastric zone. The liver and spleen are now found to be enlarged, the latter reaching some distance below the ribs. There is marked tenderness in the splenic and hepatic areas. General muscular pain and soreness are prominent symptoms. These pains are especially severe in the calves of the legs and are aggravated by movement and pressure. The mind is usually clear, delirium rare. Sleeplessness is a distressing symptom. Epistaxis is not common. The fever is intense, 104°–107° F. (40°–41.7° C.), and subcontinuous in type. Pulse is frequent, 110–130, of moderate fulness and tension, often quick, and sometimes dicrotic. Between the third and tenth days, commonly on the fifth or seventh, crisis occurs. The defervescence takes place in the course of a few hours and is frequently attended by profuse sweating or diarrhœa. The temperature may fall several degrees below the normal. In rare instances the crisis is attended by transient violent delirium. In feeble persons collapse may occur. The urine not infrequently contains albumin.

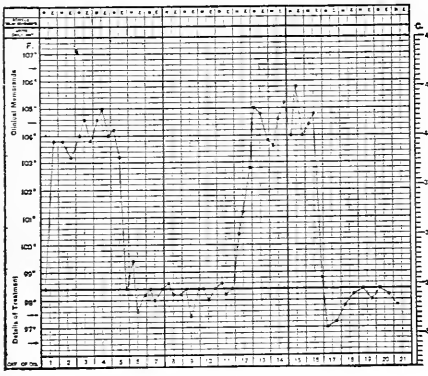


Fig. 217.—Relapsing fever.

The Intermission.—The crisis nearly always sets in during the night. The patient in the course of a few hours experiences remarkable relief. The pains, headache, and gastric symptoms promptly disappear with the fever. There is first a feeling of weakness but strength augments from day to day. This period usually lasts about a week. In some instances it does not exceed four or five days. In a limited number of cases the relapse does not occur. The Relapse.—Between the twelfth and twentieth days from the beginning of the attack, but commonly on or about the fourteenth day, the patient, with the same suddenness as before, again falls ill. The relapse sets in usually at night. The symptoms are a repetition of those of the primary paroxysm but less severe and the relapse is of shorter duration, usually about three days, sometimes not more than twenty-four hours. Occasionally a second relapse, less frequently a third, occurs and in very rare instances a fourth has been noted. Convalescence.—The close of the attack, especially after repeated relapses, finds the patient much prostrated. There is marked emaciation, and the convalescence is tardy, weeks often elapsing before the health is restored.

Complications and Sequels.—Mild bronchitis is not uncommon. Pneumonia has been frequent in some of the epidemics. Chronic valvular disease and myocarditis render the patient liable to sudden death from syncope. The sudden swelling of the spleen may cause rupture of its capsule. Nephritis is a rare complication. Hæmatemesis and hæmaturia have been noted. Parotid bubo has been a prominent complication in some epidemics. Pregnant women almost invariably abort or miscarry during the course of relapsing fever. This accident exceptionally occurs in the first paroxysm, commonly in the second. The fœtus even at the approach of term perishes and the life of the mother is often lost. Forms of ophthalmia have been common sequels in some of the epidemics. Palsies may occur as the result of peripheral neuritis.

Diagnosis.—The direct diagnosis rests upon the abrupt onset, high temperature, enlargement of the liver and spleen, the critical defervescence at the end of five to seven days, and the occurrence of relapse. Spirochætæ in the blood render the diagnosis positive. Lowenthal used the serum test upon active spirilla with positive results in 30 out of 39 tests. In 14 cases he was able by this method to make a diagnosis after the disease was past, thus determining the true nature of a previous illness of doubtful character.

Prognosis.—The prognosis is, as a rule, favorable. The mortality in different epidemics varies between two and four per cent. Death may occur from exhaustion, at the relapse, after repeated relapses, or from collapse at the time of crisis.

IV. THE VARIOLOUS DISEASES.

Variola—Smallpox.

Definition. — An acute, infectious, endemic and epidemic disease, highly contagious, characterized by fever of typical course and a general eruption which passes through the progressive stages of macule, papule, vesicle, pustule, and crust.

Etiology.—Predisposing Influences.—Smallpox may be regarded as the prototype of contagious diseases. It is endemic and in the absence of vaccination occasionally epidemic in every climate and among all races. Outbreaks are more common in the great centres of population, but when the disease is transported to countries in which it has never or not recently prevailed, as in Iceland or in North America among the aborigines, it has raged as a veritable scourge. The negro races are peculiarly susceptible and suffer more severely than whites.

Age confers no immunity. The fœtus in utero may develop the disease if the pregnant mother has contracted it. Miscarriage as in other grave infections is liable to occur and the fœtus may be born with the signs of the disease or the child at term may develop it within the period of incubation. In rare instances the fœtus may bear the scars. Sometimes the child in a smallpox hospital is born without signs of the disease, and may, if at once vaccinated, escape. Such children are very delicate. Welch has seen a case at the age of eighty-three. Sex is without influence. The menstrual period and pregnancy are supposed to

render individuals especially liable to contract the disease. Questions relating to vaccination and revaccination and exposure render generalizations in regard to these conditions useless. Previous disease has no influence. Neither acute nor chronic affections confer immunity, save in so far as patients suffering from acute infections such as scarlet or enteric fever, measles, or influenza are less exposed to the contagion in limited epidemics than persons going about.

The Exciting Cause.—Bodies resembling protozoa in the lesions were first described by Guarnieri—*Cytoryctes variolæ*. Later Councilman and his associates demonstrated a protozoon with a cytoplasmic stage and a double cycle and small structureless bodies in the lower layer of the epithelial cells. Various observers have confirmed these findings. These organisms bear a definite relation to the lesions and the hypothesis that they are the cause of the disease is tenable.

The infecting principle is thrown off in the expired air and in the exhalations from the skin, in the secretions and excretions, and in the crusts of the unruptured and ruptured pocks formed during desiccation. The disease is transmissible during the whole course of the attack from the initial stage, before the appearance of the exanthem, until the dried crusts have entirely separated and the person and clothing of the patient have been disinfected. It may be communicated by approach, contact, by a third person himself immune, and by any articles serving as fomites. The dried scales and pus and the discharges from the nose and mouth floating in the air as dust play the chief rôle in the dissemination of the virus, and it is by this means that transmission through the atmosphere, in the absence of any communication, has taken place at distances of one hundred metres or more. It is inoculable by means of the lymph of the vesicles, pus, crusts, and the blood—the contents of the vesicles being most virulent at the time when turbidity appears, the blood during the early stages of the attack. The corpses of those dead of variola communicate the disease to susceptible persons not only in the performance of autopsies or dissections but also in their ordinary disposal for burial. The danger is greatest in the immediate proximity of cases, but under certain conditions it extends to remote distances. In this connection the part played by flies and other insects is not to be overlooked. The poison is not only virulent, it is also in the highest degree tenacious and persistent. Infected clothing that has been packed away may after several years give rise to the disease and thus become the unsuspected cause of outbreaks in localities long free from the disease. Cases have been traced to baled rags brought from distant countries as an article of commerce. It clings to articles of furniture, carpets, and rooms, and is liable in times of epidemics to infect cabs and other public conveyances.

The usual mode of access is by way of the inspired air. The susceptibility to the disease is in the absence of vaccination almost universal. Natural immunity has, however, been observed, and very rare instances are now encountered in which, vaccination having been unsuccessful, even when repeated, the individual has failed to contract variola upon exposure. Temporary immunity in unvaccinated persons has also in rare cases been observed. An acquired immunity results from the attack.

In most instances it is permanent. Second attacks are exceedingly rare and third attacks almost unknown. Louis XV, of France, who had smallpox at fourteen, died of a second attack at the age of sixty-four. The immunity acquired by vaccination is of variable duration, the limit of which varies between five and ten years.

Variola is transmissible to monkeys by inoculation, and among the domestic animals, the cow and horse, a local reaction takes place. The variolous disease of sheep is analogous to but not identical with smallpox in the human body.

Symptoms.—Cases of smallpox present wide variations in intensity and clinical manifestations, from a malady trifling in itself to an overwhelming illness terminating in death as early as the third or fourth day. The differences mainly but not exclusively appear at the time of the eruption, the symptoms of the stage of invasion being much more constant. For purposes of description the following scheme is convenient:

A. Variola vera—Smallpox.
   (a) V. discreta—Discrete smallpox.
   (b) V. confluens—Confluent smallpox.
   (c) V. hæmorrhagica—Hemorrhagic smallpox.
      i. Purpura variolosa.
      ii. V. pustulosa hæmorrhagica.

B. Variola modificata—Modified smallpox.
   (a) Varioloid.
   (b) Variola sine eruptione.

The period of incubation varies from five to fifteen days. In the majority of cases it is twelve or thirteen days. The incubation is apt to be shorter in the malignant forms of the disease. At the time when inoculation was practised the local reaction and constitutional symptoms frequently appeared toward the end of the third or during the fourth day. Prodromes are as a rule absent.

The course of the attack may be divided into the stage of invasion, the stage of eruption, and the stage of desiccation and decrustation.

1. **Invasion.**—The initial symptoms are acute, usually intense, exceptionally mild. There is no constant relation between the severity of this stage and the gravity of the subsequent course of the attack. The mildest varioloid may begin with violent symptoms. On the other hand, symptoms of slight intensity at the onset are not often followed by confluence or grave hemorrhagic conditions. The attack usually begins with a chill which may be repeated several times during the first twenty-four hours. In young children a general convulsion may take the place of the chill. Severe headache usually frontal, dizziness, pain in the back, and vomiting occur. The temperature rises in the course of some hours to 103°–104° F. (39.5°–40° C.) and frequently reaches maxima of 105°–106° F. (40.5°–41.1° C.). Its type during the stage of invasion, namely, until the signs of eruption begin to appear, is subcontinuous, with slight morning remissions. The respiration and pulse are accelerated, the former not infrequently reaching 30–36 per minute, the latter 120–140. The pulse may be full and bounding; in grave and malignant cases it is often

feeble and soon becomes irregular and intermittent. The skin is hot and dry, the cheeks reddened, the conjunctivæ injected. The tongue is at first slightly swollen, indented by the teeth, and covered with a thick, moist, yellowish-white fur. Pharyngitis appears early and there is pain upon swallowing. The breath is foul. Thirst, loss of appetite, and nausea sometimes leading up to repeated vomiting, accompany the fever. The nervous symptoms of the onset persist throughout this stage. Headache, dizziness, and pains in the back and limbs become even more severe. Insomnia alternates with light slumber and delirium, sometimes wandering, sometimes furious. The lumbosacral pain is excruciating and during an epidemic, when associated with high fever, headache, and vomiting, is of diagnostic importance. In severe cases occasionally there is marked precordial oppression. Physical examination of the heart and lungs yields negative results. Exceptionally a few scattered râles are heard. The area of liver dulness is not increased. The spleen is usually palpable; it may remain normal in cases of varioloid and in hemorrhagic cases. Constipation is the rule. The urine is scanty and high colored. Febrile or toxic albuminuria is common. Hæmaturia is a frequent attendant condition in *purpura variolosa*. The blood shows no characteristic changes. It does not tend to form rouleaux. There is rapid disintegration of red cells; during the fever they are normal or increased, but upon the occurrence of defervescence the number of red cells diminishes suddenly. Regeneration takes place slowly. In hemorrhagic cases the anæmia comes on quickly and is proportionate to the amount of blood extravasation. There is no leucocytosis in the mildest cases such as occur in vaccinated persons nor in the initial stages of graver cases. Leucocytosis does not appear in the absence of complications until suppuration takes place, and is due to infection by pus organisms and not to the poison of variola itself. Menstruation is excessive and if the onset of smallpox occurs toward its close the flow is increased and prolonged.

During this stage the so-called initial or accidental rashes occur. They are more common in some epidemics than others, but are encountered in from ten to fifteen per cent. of all cases. Two varieties may be recognized which differ in form, distribution, and in prognostic importance. The more common, *roseola variolosa*, is macular, suggesting the eruption of measles, though it does not present the characteristic papules nor their grouping in crescents. This rash usually appears upon the second day and disappears within twenty-four hours, never persisting after the appearance of the pocks. It comes out, as a rule, first upon the face, next upon the body, and finally in abundance upon the extremities. It is fully developed in the course of some hours and then fades somewhat more slowly. It is more common in mild than in severe cases. The second form, *erythema variolosa*, is much less common. It appears early, usually upon the first day, and may in some cases antedate the fever and other constitutional phenomena. It consists of a vivid dark crimson efflorescence, throughout which are scattered numerous purpuric spots of varying size,—*hemorrhagic erythema*. The distribution of this rash is remarkable. The regions involved constitute the "triangles of Simon," of which the first, the more common, has its base line across the abdomen, its lateral boundaries along

the inner portions of the thighs, and its apex at the knees; the second, of which there are usually two, occupy the lateral thoracic region, the axillæ, a portion of the inner surface of the arm, and extend forward upon the chest. This rash lasts until after the true exanthem appears. It gradually fades, the purpuric spots more slowly than the surrounding erythema. Within the limits of these triangles the variolous exanthem frequently comes out less abundantly than elsewhere upon the surface of the body. Some observers, as Hebra, noted this form of the initial rash more commonly in females. It is of unfavorable prognostic omen. The duration of the stage of invasion is three days. In rare cases the eruption may first appear toward the end of the second day; more rarely still, not until toward the close of the fourth day.

2. **The Stage of Eruption.**—From the time of the appearance of the exanthem the divergence of the clinical varieties begins. The essential difference between *variola vera* or true smallpox and *variola modificata* or varioloid consists in the fact that in the former suppuration takes place in the fully developed pocks, with well-marked secondary fever, while in the latter most of the pocks undergo involution from the vesicular stage without further constitutional disturbance.

A. Variola Vera.

(a) **The Discrete Form.**—About the end of the third or the beginning of the fourth day the eruption appears. It shows itself first upon the face and scalp, particularly at the edge of the hair, and in some cases upon the wrists. It spreads downward over the trunk and extremities. By the close of the third day of the eruption and sixth of the attack it is fully developed and the surface is more or less thickly covered with pocks, which are more abundant and advanced upon the face, where they first appeared, than elsewhere. Here and there are to be seen scattered individual pocks that appear later than those which surround them. The exanthem is often conspicuously copious in local areas which are submitted to habitual pressure by the clothing, as the collar, corset, or garters, or recently irritated by some application, as iodine or a sinapism. It is usually less abundant in the hypogastric region and inner surfaces of the arms and thighs than elsewhere and upon the lower than the upper extremities.

As the eruption comes out the temperature falls, the constitutional symptoms subside, and the patient feels so much better that he regards himself as convalescent.

The evolution of the pock is as follows: The macule consists of a red spot, disappearing upon pressure and varying in size from the head of an ordinary toilet pin to a split pea. The color and distribution of the rash at this period suggest measles and the differential diagnosis, especially in adults, may be difficult. Within twenty-four hours a distinct hard papule appears which feels like a shot embedded in the skin. This rapidly becomes acuminate and there develops at the summit a minute vesicle with clear contents, which gradually extends to the size of the papule and becomes tense from the increase of contained lymph. The greater number of the fully developed vesicles present a well-marked and highly

characteristic central depression—*primary umbilication.* At the centre of this depression may be found in many but not in all of the pocks a hair follicle or the duct of a sebaceous gland. Finally the clear, opalescent contents become cloudy, then opaque and yellow, the vesicle is converted into a pustule—*stage of maturation*—and with this change the umbilication disappears and the fully developed pock becomes hemispherical. The pustule is surrounded by a distinct areola several millimetres in width and the skin is slightly swollen. By the third day the pock has reached its full development and enters upon the stage of involution or desiccation. Resorption of the contents rapidly takes place, the roof of the pustule

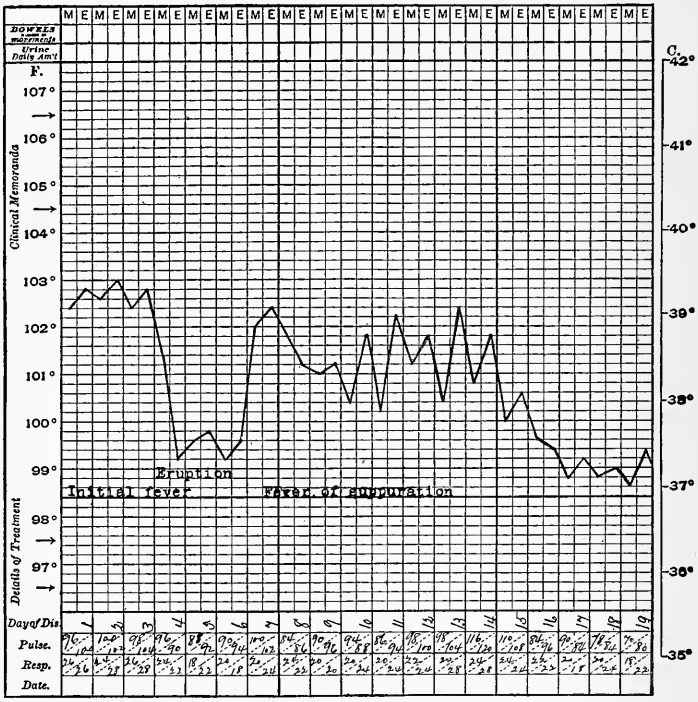


Fig. 218.—Discrete smallpox. Moderate fever of suppuration; recovery.—Royer.

sinks in,—*secondary umbilication,*—hard, yellowish-brown or blackish crusts form, which gradually separate, leaving abruptly defined, shallow scars, with glistening red bases, which in the course of time become white and finally disappear. If the cutis has been implicated permanent scars—pittings—are formed. The resulting disfigurement in ordinary discrete smallpox is commonly slight.

The time occupied by the successive stages is approximately as follows: macular one day; papular one day; vesicular three days; pustular three days; desiccation five to ten days. Puncture of the vesicles with a fine needle is followed by the escape of a portion only of its lymph, which is enclosed in several spaces limited by septa. Upon the palms and soles the pocks are as a rule scanty and owing to the thickness of the epidermis

they remain for some time deeply embedded. Early in the second week of the attack with the maturation of the pocks secondary fever and more or less severe constitutional symptoms occur. The skin is swollen, tense, and sore, especially upon the face, the eyelids are tumid, and the countenance greatly disfigured. There is marked leucocytosis. The secondary fever rises rapidly to about the range of that of the initial stadium and falls by lysis which in many cases is rapid so that defervescence is complete and the patient enters upon convalescence in thirty-six or forty-eight hours, namely, about the eleventh or twelfth day of the attack. The fever may, however, last several days. Delirium is common in severe cases and suicidal tendencies may show themselves. General septicæmia may develop.

The mucous membranes exposed to the air are involved usually at the same time with the skin; not rarely earlier. The nasal chambers, the buccal and pharyngeal surfaces, the palate, and the larynx and trachea are the seat of a more or less abundant eruption. The tongue is less frequently attacked, though now and then pocks may be observed upon the border and its under surface. The anal, preputial, and vulvar regions are later affected. The pocks upon mucous membranes are at first analogous to those upon the skin. Under the influence of heat and moisture in the vesicular stage their roofs undergo maceration and discrete superficial ulcers are formed. The mucous lesions are associated with catarrhal processes and add greatly to the sufferings of the patient. Among the symptoms to which they give rise are, in the mouth, pain and difficulty in swallowing, hoarseness and aphonia, and excessive secretion, and about the meatus urinarius in both sexes, distressing pain in micturition. These symptoms are greatly aggravated in the stage of suppuration. Among persons not protected by vaccination the discrete form has fortunately in all times been the most common.

FIG. 219.—Discrete smallpox.—Royer.

(b) **The Confluent Form.**—The pocks are closely set and run together, especially upon the face, hands, wrists, and feet. This grave form of the disease is encountered among those children and adults alike who have not been protected by vaccination and revaccination. It has not been especially common in particular epidemics nor is it transmitted from person to person. On the other hand the mildest case of varioloid may give rise to an infection resulting in *variola confluens*, while the latter may cause in a partially protected person *variola discreta* or *varioloid*. Personal predisposition must therefore enter largely into its causation.

The invasion symptoms usually are very severe. The eruption appears some twelve or eighteen hours earlier than is common in the discrete

form—in some cases by the end of the second or the beginning of the third day. The earlier its appearance the greater the danger of confluence. Its efflorescence is rapid so that by the end of the second day, the fourth or fifth of the attack, it has invaded the entire body from the head to the feet. The remarkable remission of fever and amelioration of the general symptoms seen upon the appearance of the eruption in the discrete form seldom occur. As a rule the improvement in this respect is only partial, fever persisting throughout the attack and becoming intense as suppuration takes place. The skin is swollen and hyperæmic; the individual pocks

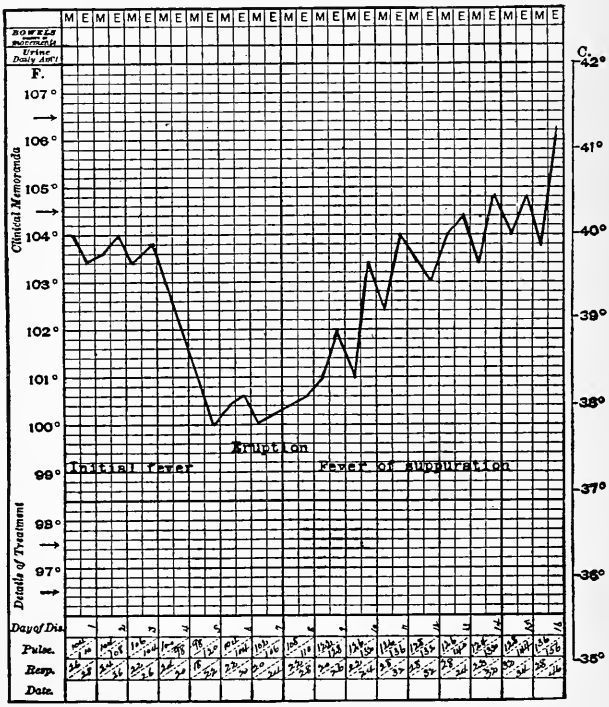


Fig. 220.—Confluent smallpox. Incomplete defervescence upon appearance of rash; severe secondary fever; death on the 16th day.—Royer.

in the papular stage are small and crowded upon the skin; they are rapidly converted into vesicles, which, increasing in size, soon become pustules. During this process confluence takes place and extensive superficial abscesses are formed. The face in severe cases presents the appearance of a thick yellowish mask. Upon the arms and legs the pocks are numerous and limited patches of confluence are sometimes seen, especially upon parts subjected to pressure, as the buttocks, while they remain discrete upon the trunk. The swelling of the hands and feet increases and these parts are the seat of most distressing tension, pain, and tenderness. The eruption is abundant upon the mucous membranes of the nose, mouth, pharynx, and larynx. Confluence may give rise to extensive superficial ulceration. Deep abscess formation may occur in the tonsils or in the retropharyngeal

tissues and necrosis of the laryngeal cartilages which may be associated with œdema of the glottis. The cervical lymph-glands are enlarged and tender. At this period purulent metastases in distant organs frequently develop. Toward the end of the first or at the beginning of the second week the fever rises to 104° F. (40° C.) or higher, the pulse to 120. De-

Fig. 221.—Confluent smallpox.—Royer.

lirium, very often maniacal, sets in; the patient is often with difficulty restrained from leaving his bed, and with the signs of a profound toxæmia, progressively feebler and more frequent pulse, subsultus, involuntary discharges, or preagonistic hyperpyrexia, death closes the scene. When recovery occurs, the cutaneous and constitutional symptoms undergo gradual improvement, and irregular fever may even in the absence of complications prolong the convalescence into the fourth week.

Fig. 222.—Exfoliation of the palmar epidermis containing embedded pocks occurring in the later stage of severe smallpox.—After Welch and Schamberg.

3. **Stage of Desiccation and Decrustation.**—The areola fades; the pustule sinks and becomes flattened; its edge is sharply defined against the surrounding skin and separation gradually takes place, the entire process occupying in V. discreta two weeks or more and in V. confluens a longer time. As a rule the desiccation begins in the face and scalp, where crusts may be seen, while upon the extremities the exanthem is still in the pustular stage. In some instances the desiccation takes place on all parts of the skin at the same time. Many of the pustules break and the exuding contents dry in the form of broad thin crusts. The process of desiccation is attended by intense itching. The pocks upon the palms and soles are limited in number and form hard circumscribed nodules in the thick epi-

dermis, which in the course of three or four weeks undergo separation and may be picked out. The hair usually falls out and in some cases the nails are lost. In the confluent form the thick epidermis of the hands and feet is sometimes cast off entire. The crusts upon separation sometimes reform and the ulcerative lesions of the skin heal slowly by granulation. Upon the face the resulting scars are much more disfiguring than the pitting of V. discreta. They are extensive, of irregular outline, and intersected by lines and bands which gradually undergo contraction, causing ectropion of the eyelids and lips and interfering with the muscles of expression.

The appearance of the patient in the stage of maturation, particularly in V. confluens, is horrible. The swollen face, thickly covered with pustules and blebs, some of which are broken and exude a sticky pus, or with a hideous mask of necrotic skin, the tumid and closed eyelids, the distorted nose and lips, the disfigured ears, the foul secretions, and the stench which surrounds the wretched being create an impression not to be forgotten and merit the popular adjective *loathsome* applied to the disease.

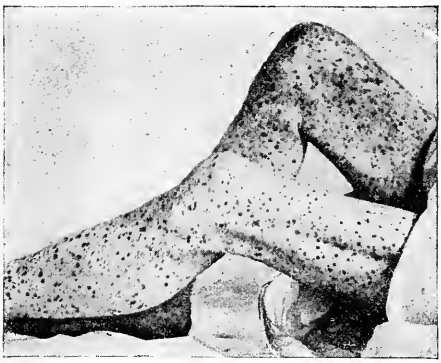


Fig. 223.—Hemorrhagic smallpox (Ross V. Patterson).

(c) **Hemorrhagic Forms.**—i. **Purpura Variolosa.**—This is the most malignant form of variola. It is fortunately comparatively rare and in some epidemics no cases have been observed. It may occur at any period of life, is less common among children than grown persons, and affects as a rule young and vigorous adults. The influence of vaccination and especially of repeated revaccination is of the greatest importance in preventing this clinical manifestation of the variolous infection. The incubation is short—five to eight days. Prodromes, especially lumbosacral pains, are not uncommon. The invasion is attended with profound constitutional disturbance. Fever may be moderate, but there is great prostration; the pulse is small and frequent, the respiration accelerated, and the patient experiences a feeling of overwhelming illness. Headache and backache are severe and precordial and epigastric distress are often associated with vomiting and purging. The mind remains clear. Commonly upon the second day, sometimes earlier, a diffuse, scarlatiniform rash makes its appearance upon the lower part of the body and the extremities and shortly thereafter upon the face. Purpuric spots of varying size rapidly appear. Ecchymoses invade and frequently entirely cover the face. The conjunctivæ, eyelids, and loose tissues adjacent are distended with a sanguinolent œdema and in a short time the greater part of the surface of the body is involved in a livid, purplish-red discoloration. Mucous hemorrhages are common, epistaxis, bleeding from the gums, and hæmaturia being the usual forms; hæmoptysis, hæmatemesis, and melæna less frequent. Metrorrhagia is common and pregnant women abort. In rare instances death may take place without the occurrence of free hemorrhage. Very

often there is no trace of the exanthem. If life be prolonged a few scattered blood-tinged papules may be discovered upon the forehead and wrists. This form of variola terminates in death within a week and very often as early as the fourth or fifth day.

ii. **Variola Pustulosa Hæmorrhagica.**—This form is much more common than the preceding and usually occurs in feeble and cachectic persons and drunkards who are not protected by vaccination. The attack develops as an ordinary severe case of variola, which becomes hemorrhagic in the vesicular or pustular stage. Exceptionally bleeding takes place into some of the pocks while yet in the papular stage. The bleeding is in many instances restricted to the eruption upon the lower part of the body and the lower extremities. Blood extravasation may also involve the skin beyond the pocks and free hemorrhages from mucous surfaces may lead up to the fatal issue. The earlier in the course of the attack the hemorrhages appear the graver the outlook. Death commonly results at the end of the first or in the beginning of the second week. Recovery may occur in cases in which hemorrhage into the pocks does not take place until the stage of suppuration. A distinction must be made between this form of variola and cases of V. discreta in which, owing to mechanical violence, pressure, or other accidents, blood is extravasated into a few vesicles or pustules.

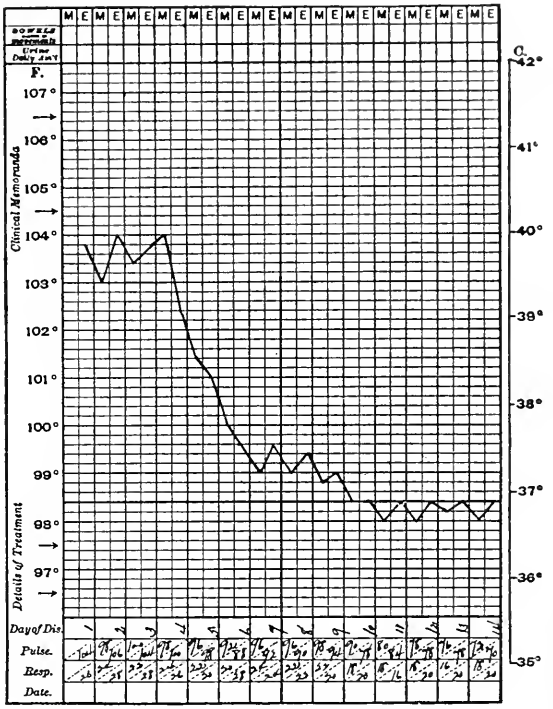


FIG. 224.—Severe varioloid.—Royer.

To the latter group must be referred cases of hemorrhage into the pustules of the legs in patients who have gotten out of bed during their delirium. Osler describes a series of six cases in which hemorrhage into the vesicles was followed by "a rapid abortion of the rash and speedy recovery."

B. Variola Modificata.

(a) **Varioloid.**—This term is applied to the modified form of smallpox which occurs in persons who possess a partial immunity as the result of vaccination and revaccination. It is characterized anatomically by the fact that the typical exanthem causes as a rule only superficial lesions

upon the skin and therefore rarely leaves permanent scars; clinically by the absence of the stage of suppuration and the secondary fever and a shorter and more favorable course. The period of incubation is the same. The onset is abrupt and in many cases the initial symptoms are severe. Generally the symptoms of the stage of invasion are relatively mild. Headache and backache may, however, be intense. If an initial rash appears it is the macular variety—*roseola variolosa*. The papules appear toward the close of the third or in the beginning of the fourth day, first upon the face and then elsewhere, spreading from above downward. They vary in number from ten or twelve upon the face and hands to a copious eruption distributed over the entire surface. Within forty-eight hours their appearance is complete, although during the latter part of this period a few fresh papules may be recognized here and there among those which first appeared. The fever ends at once by crisis and the general symptoms rapidly improve. The development of many of the pocks is arrested in the papular stage; others abort early in the vesicular stage and in some few the contents of the vesicles may become cloudy and slightly opaque. Desiccation sets in early and goes on with rapidity. Convalescence is usually complete.

(b) **Variola sine Eruptione.**—During outbreaks of variola cases are sometimes observed in which no trace of eruption can be discovered. The diagnosis rests upon etiological as well as upon clinical grounds. A history of exposure, sudden onset, fever, intense lumbosacral pains, and critical defervescence upon the third day justify a provisional diagnosis of smallpox without eruption. The transmission of the disease to others would render the diagnosis positive. Variola sine eruptione occurs in young persons who have been well vaccinated and invariably runs a favorable course.

The modification of variola caused by artificial inoculation is no longer encountered in western countries. About the eighth day local reaction was manifest at the point of inoculation. Fever and constitutional symptoms developed and were followed by a typical exanthem, not usually copious.

A rare anomaly in the pock is described under the name of horn-pox or wart-pox—*V. verrucosa*. The eruption appears upon the third or fourth day but instead of developing as usual the papules undergo desiccation upon the fifth or sixth day and are converted into dense warty or horny nodules. This change is more common upon the face than elsewhere.

Complications and Sequels.—The complications are not numerous and mostly develop during the stage of suppuration. They consist mainly of extensions of the suppurative inflammatory process in the skin or mucous membranes or of metastatic infections. It follows that they are more common and severe in proportion to the extent and intensity of the suppuration—in V. confluens than in V. discreta, and infrequent in varioloid. Bed-sores and acute gangrene are frequent in severe cases. Erysipelas is not uncommon. Phlegmonous inflammation of the skin may occur. Furunculosis and acne are often troublesome during convalescence. Superficial erosions in the larynx may in healing give rise to adhesions which result in permanent hoarseness; the cartilages may be involved and acute œdema of the larynx may cause sudden death. Lesions of the larynx play an important part in the causation of bronchitis and bronchopneumonia which is perhaps the most common of the complications. Croupous

pneumonia is infrequent; pulmonary abscess may occur. Purulent pleurisy has been common in some epidemics. Cardiac complications are infrequent. Myocardial changes are observed. An apex systolic murmur may occur. Pericarditis is rare. Simple endocarditis is not common in smallpox. Malignant endocarditis has been in some instances found post mortem. Venous thrombosis may occur during the later course of the attack. In the digestive tract parotitis and inflammatory affections of the other salivary glands occasionally occur. Their frequency varies in different epidemics. Pseudomembranous angina is common in severe cases and especially in the hemorrhagic forms. The vomiting of the initial stage is not apt to persist. Diarrhœa is frequent, especially in children. Dysenteric symptoms are

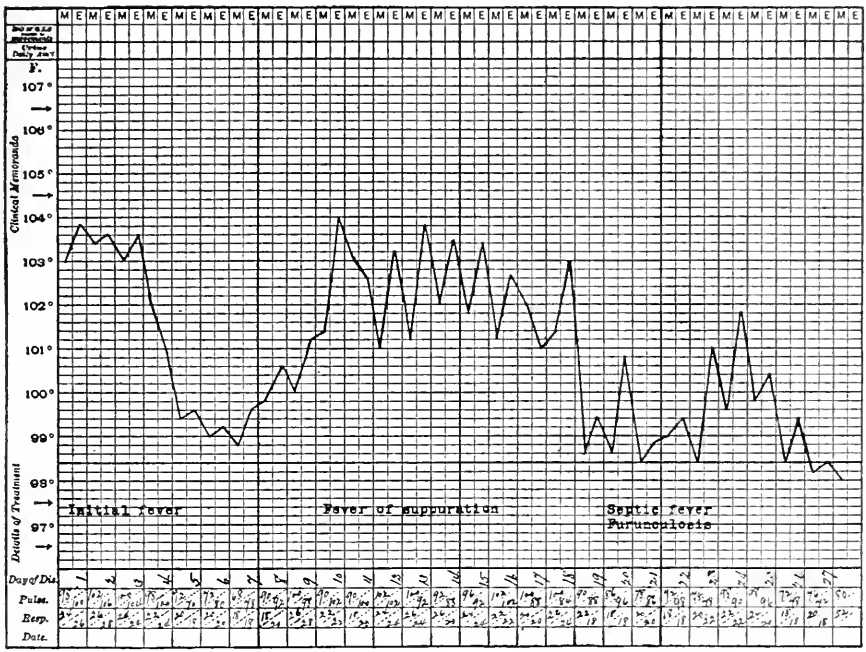


FIG. 225.—Confluent smallpox; furunculosis as a sequel.—Royer.

frequently mentioned by the older authorities but are rare at present. Complications in the urinary tract are infrequent. Toxic albuminuria is common; nephritis rare. Hæmaturia is usual in the malignant cases. Inflammation of the ovaries and of the testicles—orchitis variolosa—may occur. Pregnant women usually abort. Pyæmic arthritis and acute necrosis of bones or cartilages may, as in the other severe acute infections, develop during the convalescence. Diabetes mellitus is a rare sequel.

The nervous system is especially prone to react to the variolous infections. In children general convulsions are common at the outset and may be repeated. In adults and especially in alcoholics maniacal delirium may terminate in coma. Post-febrile insanity sometimes occurs. It is less common than after influenza and enteric fever and commonly ends in recovery. Epilepsy is a very rare sequel. Purulent meningitis and enceph-

alitis are likewise rare complications. Hemiplegia has been observed in a few instances. Transient aphasia has been noted. Paraplegia may occur at any time during the attack. It begins abruptly, is usually subacute, and may involve the sphincters. In some instances the symptoms have been those of acute ascending paralysis and the termination rapidly fatal. The palsies of the lower extremities and the monoplegias observed occasionally are manifestations of an infectious neuritis. Ataxic symptoms may occur. Paralysis of the soft palate analogous to that which follows diphtheria sometimes occurs. Paralysis of individual muscles or muscle-groups, as the deltoid, and circumscribed areas of cutaneous anæsthesia have been described.

Otitis media purulenta is a frequent complication. It results from the extension of the catarrhal inflammation by way of the Eustachian tube. It usually develops during the stage of suppuration. Exceptionally purulent disease of the mastoid arises with necrosis of the bone, or the labyrinth may be involved. Serious affections of the eye with resulting blindness were common in former times. They are relatively infrequent at present because of the greater attention now given to the early ocular lesions. The conjunctivitis incident to the disease may become chronic. Diffuse keratitis may occur and result in ulceration and perforation with destruction of the eyeball. Iritis and choroiditis are less common. In the purpuric cases retinal hemorrhages may occur. Pocks develop very commonly upon the outer surface of the eyelids and result in scar formation with ectropion and its attendant evils. Much less commonly they involve the palpebral conjunctiva and very rarely the ocular conjunctiva. Synechiæ may result.

External and internal nasal deformities sometimes result from the ulcerative processes and subsequent scar formation. Necrosis of the cartilaginous septum with perforation, occlusion of a nasal chamber, partial adhesion of the soft palate, and loss of the sense of smell are among the sequels of smallpox.

Diagnosis.—A correct diagnosis where there is a question of variola is one of the most critical and important of the duties of the physician. A mistake may be the cause of an extended and disastrous epidemic.

The DIRECT DIAGNOSIS of typical variola is unattended by difficulty after the appearance of the eruption. In the atypical and modified forms the difficulties are frequently insurmountable. During the prevalence of an epidemic every case of sudden illness is suspected. The sudden occurrence of headache, rigors, intense backache, epigastric pain, nausea and vomiting, and high temperature is important. The initial rashes may be misleading. The measly rash—*roseola variolosa*—bears in many instances some resemblance to measles, for which the disease may be mistaken. More commonly, however, during an epidemic measles is mistaken for variola. The scarlatinal rash—*erythema variolosa*—lacks as a rule the vividness of the eruption of scarlet fever and differs from it in distribution, occupying the abdominocrural or pectoral triangles of Simon. In fact the occurrence of this rash in connection with the foregoing symptoms renders the diagnosis of smallpox almost positive. A history of exposure and the absence of vaccination scars are of diagnostic importance. The

development toward the close of the third or at the beginning of the fourth day of an eruption of red macules upon the face, scalp, and wrists, rapidly becoming papular and feeling like shot embedded in the skin, coincidently with a marked remission in the febrile movement, renders the diagnosis positive. In any doubtful case, especially in the negro, the buccal and faucial mucous membrane should be carefully examined.

In malignant hemorrhagic smallpox death may occur before the characteristic rash develops. If the patient survive to the end of the third or fourth day a few shrunken, shotty papules may be felt about the roots of the hair and upon the wrists.

Differential Diagnosis.—In the initial stage there are points of superficial resemblance to the following diseases: 1. Scarlet Fever.—Erythematous sore throat, more or less painful. The rash is brighter and more intense than the erythematous initial rash of smallpox, in which petechiæ are very commonly present. It appears first upon the chest and throat and is rapidly diffused. Eosinophilia is marked. 2. Measles.—In the stage of invasion, marked catarrhal symptoms and Koplik's spots. The rash appears about the fourth day but is maculopapular, lacking the firmness of the variolous papule and showing a crescentic arrangement. There is no remission in the febrile movement upon the development of the rash. An early leukopenia occurs. 3. Typhus.—The eruption is rare upon the face and when present in this region is comparatively faint. The macules are not hard or elevated. There is no fall of temperature upon the appearance of the eruption.

During the vesicular and pustular stage smallpox may be mistaken for: 4. Varicella.—Errors in diagnosis between these two diseases are very common. In varicella initial symptoms are usually absent. The eruption is always discrete and appears in successive crops. It is usually not observed until it has reached the vesicular stage. The vesicles are irregularly oval or circular, tensely distended with a transparent fluid. They are clear, bright, and pearly. Primary umbilication does not occur, but in rare cases, as the contents of the vesicles undergo resorption, the roof of the vesicle falls in, giving rise to the condition known as secondary umbilication. 5. Pustular Syphilides.—Individual pustules occasionally bear a close resemblance to the variolous pock. The polymorphous character of syphilitic eruptions, their symmetrical distribution, their persistence, the presence of mucous patches, the history of the case, and the nature of the febrile movement when present serve to render the differential diagnosis a relatively easy matter. 6. Pustular glanders is attended with malaise, pyrexia, and pain in the limbs. There is an eruption of indurated red papules upon the summit of which pustules develop. There is a fetid nasal discharge. The invasion symptoms and the course of the disease are totally unlike smallpox. 7. Cerebrospinal Fever.—The sudden onset, intense symptoms, and petechial rash may lead to errors of diagnosis. Painful rigidity of the back of the neck and spastic contraction of the limbs may be present in both these conditions. Papules should be carefully sought for at the roots of the hair and upon the wrists. Photophobia, pupillary inequalities, strabismus, and very irregular fever suggest meningitis rather than smallpox. Lumbar puncture is important. 8. Drug Exanthems.—Iodide of potassium, the bromides, and the local

use of croton oil may produce rashes suggesting smallpox, but the resemblance is extremely superficial and the true nature of these affections becomes apparent upon examination.

Prognosis and Mortality.—In persons not protected by vaccination smallpox is a very fatal disease. In the older epidemics the mortality ranged from 40 to 60 per cent. and smallpox was dreaded alike from its loathsomeness, its high mortality, and its serious sequels. At the present time the mortality of smallpox is greatly influenced by:

1. Vaccination and Revaccination.—During the nineteenth century smallpox epidemics diminished in the most remarkable manner in extent and frequency and showed a correspondingly lowered mortality. Nevertheless among the unvaccinated the gravity of the disease and the death-rate are practically unchanged. MacCombie's statistics show in 3940 unvaccinated cases of all ages 1758 deaths—44.6 per cent. Among 17,756 vaccinated cases 1441 deaths, a mortality of 8.1 per cent. Welch, Municipal Hospital, Philadelphia, reported in 2831 cases of variola 1534 deaths, a mortality of 54.18 per cent., and in 2169 cases of varioloid 28 deaths, a mortality of 1.29 per cent. In Sheffield in the outbreak of 1887–8, of 4703 cases 474, or 10 per cent., terminated fatally. Of 552 patients not vaccinated 274, or 49.6 per cent., died; while of 4151 vaccinated patients 200, or 4.8 per cent., died.

The character of the vaccination is of great importance as affecting the prognosis. The relative value of multiple or repeated vaccinations is shown by McCombie's analysis of 11,724 cases. This author regards an area of $\frac{1}{3}$ to $\frac{1}{2}$ square inch of well-foveated surface as indicating efficient vaccination. He found the mortality among cases with one good mark to be 6.4 per cent.; among those with one indifferent mark, 16.7 per cent. Among those with two good marks, 3.7 per cent.; among those with two indifferent marks, 11.2 per cent. Among those with three good marks 3.7 per cent.; among those with three indifferent marks 7.4 per cent. With four or more good marks 2.7 per cent.; with four or more indifferent marks 4.8 per cent., and concludes that the protection against a fatal attack is three or four times greater among patients with efficient than those with indifferently successful vaccination. W. M. Welch analyzed 5000 cases with reference to the character of the cicatrices, whether produced in primary vaccination or in revaccination, with the following result: The mortality among persons having good scars was 8 per cent.; fair scars 14 per cent.; those with poor scars 27 per cent.; average mortality in persons showing the cicatrices of vaccination 16 per cent.; mortality among unvaccinated persons 58 per cent.

Death from smallpox is rare in persons whose primary vaccination was efficient and in whom revaccination has been successful. The above well-established facts are of the greatest practical importance.

2. The Virulence of the Attack.—Smallpox modified by efficient vaccination and revaccination is a comparatively trifling disease with a death-rate but little exceeding 1 per cent. of all cases. Ordinary discrete smallpox is a grave affection with a greatly increased mortality. Confluent smallpox is even more grave, the majority of the cases terminating in death, and those recovering frequently suffering from serious, often

irremediable, sequels. Finally, the hemorrhagic form—*purpura variolosa*—is invariably fatal. Petechial rashes and hemorrhagic phenomena are especially unfavorable. A rise of temperature directly after the appearance of the eruption is a bad sign. Continuing delirium, persistent high temperature, and convulsions are of grave prognostic omen.

3. The Patient's Surroundings.—Unfavorable hygienic conditions, overcrowding, poverty, and want greatly increase the mortality.

4. The Occurrence of Complications.—The complications of variola affect the prognosis unfavorably. Certain epidemics have been attended with an unusual death-rate in consequence of the frequent occurrence of ordinary grave complications. The laryngeal and pulmonary complications are especially ominous.

5. Age.—Among the conditions unfavorably affecting the death-rate in individual cases age is of great importance. In young children the disease is peculiarly fatal. Unvaccinated infants in the first year mostly die, and the mortality is high up to the tenth year. From ten to twenty years of age there is a slight decrease in the death-rate, which after the thirtieth year again rises.

6. Previous Illness.—Previous severe illness and alcoholism render the prognosis unfavorable.

7. Pregnancy.—Pregnant women are especially liable to the disease in the confluent and hemorrhagic forms. Abortion usually occurs and is apt to be followed by septic infection. To this rule there are fortunately occasional exceptions. The fœtus may show a well-developed eruption and quickly die or it may develop the eruption shortly after birth. In cases in which abortion does not occur the child may undergo the disease in utero and be after birth immune alike to vaccination and variola.

Vaccinia, Cowpox, Kinepox.

Definition.—An eruptive disease of the cow, communicable only by inoculation and causing, when transmitted to the human being, local reaction in the form of a pock and constitutional disturbances which are followed by a more or less lasting immunity against smallpox.

Vaccination.

Definition.—The artificial inoculation of vaccine virus for the purpose of producing immunity against smallpox.

Arm to arm vaccination was formerly very generally practiced in order to perpetuate the lymph and secure its greatest purity. The use of crusts came into vogue at a later period. Bovine vaccine lymph has now come into general use, and has the advantage over the arm to arm method of avoiding the opening of the vesicle and thus affording the opportunity for accidental infection and of wholly eliminating the danger of syphilis and other infections. When it is necessary to use human lymph it should be taken upon the eighth day from a typical unbroken vesicle in a perfectly healthy child at least three months old. The vesicle must be pricked at several points, care being taken not to draw blood. The lymph may be

in a preserved dry state upon sterilized bone points or slips. When required for use the dry lymph is moistened by a few drops of warm sterilized water. It may also be preserved in capillary glass tubes, each containing the quantity required for one vaccination, sufficiently long to admit of sealing in the flame of a spirit lamp, thin enough to enable them to be instantaneously sealed, and strong enough to be handled and transported.

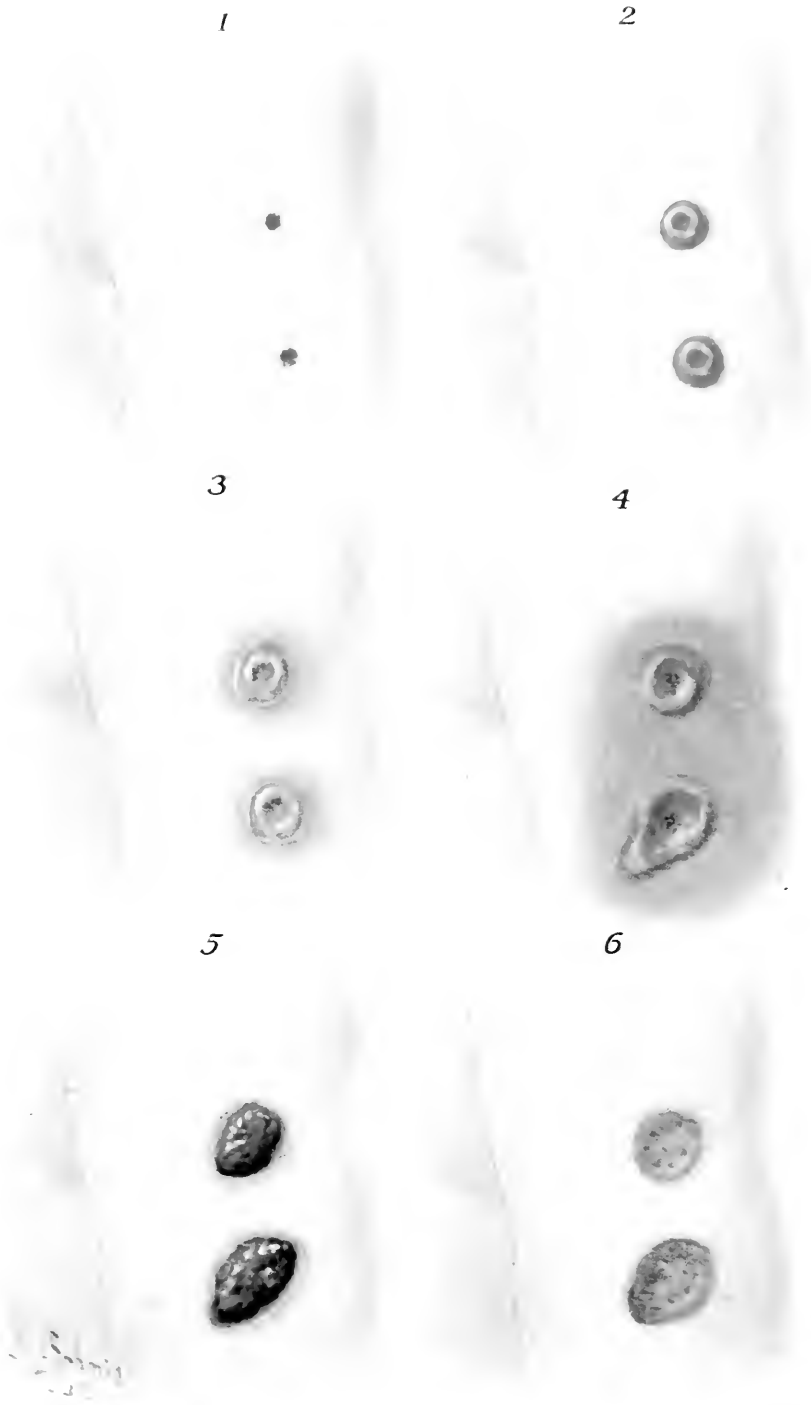
Glycerinated Lymph.—The thorough incorporation of four parts of a sterilized 50 per cent. solution of chemically pure glycerin in water with one part of lymph or vesicle pulp, and the storing of this mixture in sealed capillary glass tubes, protected from light for some weeks, is followed by the destruction not only of the ordinary saprophytic bacteria found in the lymph, but also of tubercle bacilli and the streptococcus of erysipelas. Lymph thus treated is fully as efficient as ordinary lymph.

Fig. 226.—Scars from an infantile vaccination.—After Welch and Schamberg.

Vaccinia produced by humanized lymph has a somewhat more rapid evolution than that caused by bovine virus and is attended with milder constitutional symptoms.

The Technic.—The outer surface of the arm near the insertion of the deltoid is usually selected. In infants the left arm is preferable. In females the outside of the leg just below the knee is sometimes chosen. The surface must be washed, dried with a soft towel, and the lymph inserted by puncture, multiple superficial crossed incisions, or after the removal of the epidermis by scraping. The spots are to be rendered moist by the exuding serum but care should be taken not to draw blood. For this purpose a thoroughly sterilized old-fashioned thumb lancet or an ordinary flat-headed surgical needle should be employed. The insertion should be performed at two points about an inch apart and the diameter of the abraded or scarified area should be about one centimetre. The clothing should not be replaced until the serum has thoroughly dried. A thin layer of sterilized gauze should be lightly applied and held in place by means of adhesive plaster, not encircling the limb. This should be occasionally renewed. The pock should be kept dry and clean, and may be lightly dusted with starch or toilet powder. The newborn should be vaccinated only during the prevalence of smallpox. Children are commonly vaccinated in the course of the third month. In case of failure the operation must be repeated. Persons exposed to the contagion of smallpox should be immediately revaccinated. The immunity conferred diminishes with time. Revaccination should be performed at the seventh year of age, again at puberty, and from time to time as epidemics occur.

Typical Vaccination.—The period of incubation varies from three to five days. At the end of this time local reaction shows itself in the form



TYPICAL VACCINATION.

1. Papules. 2. Vesicles with clear contents. 3. Vesicles with opaque contents. 4. Fully developed pocks with erythematous areola. 5. Crusts. 6. Appearance immediately after separation of crusts.

of one or more reddish papules at the point of inoculation. These in the course of five days develop into compound vesicles, the contents of which are at first clear but later become opaque. By the eighth day the vesicle is fully developed and is round or oval with prominent and well-defined edges and a depressed or umbilicated centre. About the tenth day an erythematous areola usually appears and the contents of the pock become purulent. The surrounding skin is swollen, indurated, and tender. Scabbing now begins at the centre of the pock and rapidly extends toward its borders. The areola fades about the end of the second week, and the pock

FIG. 227.—Two sisters suffering from smallpox. The one on the right was successfully vaccinated in infancy; she contracted a mild varioloid and recovered without any scarring. The other, unvaccinated, developed a severe smallpox, and recovered, though considerably pitted.—After Welch and Schamberg.

is converted into a thick brownish crust which gradually becomes dry and hard and separates between the twentieth and twenty-fifth days after the vaccination, leaving a scar of a dusky red color which gradually becomes white and pitted or foveated. The corresponding superficial lymphatic glands, namely, the axillary or inguinal, as the case may be, during the evolution of the pock become slightly enlarged and tender.

The constitutional reaction is commonly slight. It shows itself by moderate fever, restlessness at night, loss of appetite, and irritability. These phenomena usually appear upon the third or fourth day and continue until the early part of the second week. Erythema, roseola, or urticaria may develop at any time during the course of the vaccine disease. These eruptions are usually transient. Leucocytosis shows itself about the third day coincidently with the appearance of the local eruption, and

again about the time the pock reaches maturity. The resulting immunity against vaccinia like that against variola varies in duration in different individuals. In rare instances it is permanent, but as a rule successful revaccination may be performed in the course of some years. The pock of revaccination, however, lacks in most instances the typical development of the primary vaccine lesion. The constitutional reaction in revaccination is sometimes severe. If no characteristic lesion follows the attempt at revaccination, the operation should be repeated once or twice at short intervals.

Atypical Vaccinia in Man.—1. Variations in the Number of Pocks.

(a) *Supernumerary pocks* occasionally develop in the vicinity of the original vaccine lesion.

(b) *Confluent pocks* may in rare instances be formed by the coalescence of the supernumerary pocks either among themselves or with the original lesions.

(c) *Generalized vaccinia or vaccinal eruptive fever* is less common. It consists of a vaccine rash developing in various parts of the body, especially about the wrists or on the back. Secondary pocks usually begin to develop about the eighth or tenth day after vaccination and are often more abundant on the vaccinated limb than elsewhere. The pocks appear in successive groups so that they may be seen in all stages of development. The disease sometimes lasts several weeks.

(d) *Vaccinal Eruptions Generalized by Autoinoculation.*—Supernumerary pocks may be produced by scratching with the nails after they have been in contact with the ruptured vaccine pock. They may occur in any part of the body and vary from one or two to many. The number is sometimes very great. They have been observed upon the cheek, lips, tongue, buttocks, breast, and the genital organs. On the mucous surface of the vulva the resulting ulceration may give rise to the suspicion of venereal disease.

(e) *Local vaccinal eruptions* may arise at the seat of previously existing cutaneous lesions, as impetigo, eczema, acne, or psoriasis.

2. Variations in the Size of the Pocks.

(a) *Two or more of the primary vesicles* caused by vaccination may coalesce to form one large pock.

(b) *The size of the pock* may be increased by coalescence of supernumerary pocks in the immediate neighborhood.

3. Variations in the Contents of the Pock.—In cachectic individuals the contents of the vesicle, instead of being clear and limpid at the end of the first week, may be watery, hemorrhagic, or purulent.

4. Variations in the Evolution of the Pock.

(a) *Acceleration.*—The pock develops more rapidly in summer than in winter. Its evolution is apparently hastened by idiosyncrasies on the part of individuals and by the character of the lymph employed.

(b) *Retardation.*—Cases occur in revaccination in which vesicles apparently aborted become active a week or more after the original insertion.

(c) *Abortion.*—The non-development of the pock is determined by the immunity of the patient, the quality of the lymph, and the skill of the vaccinator. In revaccination a bright red papillary lesion, "raspberry excrescence," sometimes develops about a week after the insertion of the

lymph. Vesicles do not form and the papules remain hard and dense for several weeks. There is no areola, and healing ultimately takes place without the formation of a scar.

5. Variations in the Involution of the Pock.—These anomalies are determined by vaccinal injuries. Secondary infection may take place at the time of the operation or subsequently if the pock is injured. It may be due to the use of contaminated lymph or infected instruments or may arise at a later period from other causes. Vaccination is not wholly free from the danger of accident. Severe inflammation, suppuration, deep-seated ulceration, or gangrene may occur in mismanaged cases. Erysipelas is an occasional complication. Cellulitis, abscess formation, and septicæmia may occur. These accidents are, however, not peculiar to vaccination. They may arise in any lesion of the skin in default of proper antiseptic measures.

6. Variations in Healing and the Formation of Scar.—The lesion of the skin caused by vaccination is usually fully healed by the end of the third week. It may, however, remain open for some weeks. The scar sometimes manifests hypertrophy or puckering or runs into keloid.

7. Transmission of Chronic Specific Disease by Vaccination.

A. *Vaccination and Syphilis.—Vaccinosyphilis.*—Syphilis has been transmitted by vaccination, but the number of well-authenticated cases is limited. The general use of bovine lymph has rendered invaccinated syphilis a remote possibility. It may, however, occur in consequence of the use of lymph taken from an individual suffering from syphilis or from contamination of the instrument or wound or it may be due to infection from the vaccinator. The sequence of events is as follows:

If the subject be susceptible to vaccination the pocks may show no departure from the normal course, but in some instances they abort. If they be irritated, the vaccinal sore may become inflamed, suppuration may occur, and the ulcers may scab over and again break out. Whether the vaccination runs a typical or an atypical course, a chancre with indurated base eventually forms at the point of inoculation, and the signs of general infection at a later period.

Acland makes the following deductions from a well-known case of autovaccination with vaccine virus from a syphilitic child; they are in entire accordance with general observation:

(a) That vaccination can be successfully performed with lymph taken from a source tainted with syphilis without necessarily communicating that disease.

(b) That if syphilis be communicated in the process of vaccination it does not follow that all the points of insertion will become infected.

(c) That the evolution of syphilis, as regards the primary and secondary stages, is not necessarily disturbed; that it is neither accelerated nor retarded by simultaneous vaccination.

(d) That no care in the selection of lymph obviates the risk of vaccinating from an obviously tainted source.

(e) That when syphilis is communicated by vaccination, the first appearance of the disease is at the seat of puncture.

B. *Vaccination and Tubercle.*—The communication of pulmonary tuberculosis as a result of vaccination is of exceeding rarity. It may in

fact be doubted whether it has ever occurred. In well-regulated vaccine laboratories the animals used are previously submitted to the tuberculin test. It has been suggested that they should, after the collection of the vaccine material, be slaughtered and submitted to examination for tuberculous lesions. In case of their presence, the vaccine material must be rejected.

In some few instances lupus has been observed at the seat of vaccination.

C. *Vaccination and Leprosy.*—The alleged cases of transmission of leprosy by vaccination are open to serious doubt.

D. *Vaccination and Cancer.*—There are no authentic cases on record in which cancer has resulted from vaccination or developed in the vaccination scar.

E. *Vaccination and Tetanus.*—The vaccine lesion, like other wounds of the skin, renders the patient liable, under certain circumstances and the absence of proper precautions, to tetanus infection. A limited number of instances of fatal tetanus after vaccination are reported.

Acland has arranged the dates at which various eruptions or complications may be looked for after vaccination, as follows:

1. During the first three days: erythema; urticaria; vesicular and bullous eruptions; invaccinated erysipelas.

2. After the third day and until the pock reaches maturity: urticaria; lichen urticatus; erythema multiforme; accidental erysipelas.

3. About the end of the first week, and generally after the maturation of the pocks: generalized vaccinia—(a) by autoinoculation, (b) by general infection; impetigo; accidental erysipelas; vaccinal ulceration; glandular abscess; septic infections; gangrene.

4. After the involution of the pocks: invaccinated diseases, for example, syphilis.

V. VARICELLA.

Chicken-pox.

Definition.—An acute infectious, endemic and epidemic disease of childhood characterized by mild constitutional symptoms and a vesicular exanthem which develops in irregular, successive crops.

Chicken-pox was formerly confused with smallpox and until recently there have been those who regarded it as a greatly modified and very mild variety of smallpox. It is now generally looked upon as an entirely distinct disease. One of these diseases never gives rise to the other; the attack of one does not confer immunity against the other, and it is no rare event for a person who has recently suffered from one to contract the other. Vaccination confers no immunity against varicella, and children who have recently suffered from varicella react to vaccination in the ordinary manner.

Etiology.—Predisposing Influences.—Varicella is a wide-spread disease, endemic and frequently epidemic in the great centres of population, usually in the autumn or early spring. As in the case of other readily transmissible infections, sporadic cases occur and frequently become the centre of house epidemics or extended outbreaks. The liability is general, and scarcely any individual who has not had the disease escapes when it makes its appearance in a school or other public institu-

tion. It is a disease of childhood, the majority of cases occurring before the eighth year and few after the tenth. It is comparatively infrequent during the first year and, though rare, occasional cases occur after puberty. Sex is wholly without influence as a predisposing factor.

The exciting cause is not known. The disease is highly contagious and usually communicated in the ordinary intercourse of children in the family, the school, or the playground. Direct contact is not necessary, the infection being communicable at some little distance by the air and to greater distances by persons who pass from the sick to those who are susceptible, and finally by means of fomites. Inoculation experiments have shown that it is present in the contents of the vesicles. Outbreaks of varicella are sometimes associated with measles, whooping-cough, scarlet fever, or variola.

Symptoms.—The period of incubation is usually thirteen or fourteen days Prodromes as a rule do not occur. In a majority of the cases the eruption is the first sign. It first appears usually upon the face and spreads into the hairy scalp and progressively over the trunk and extremities. It sometimes comes out first upon the back and shoulders and very often at the same time upon the wrists and forearms. The pocks are more numerous upon the trunk and upon the upper than the lower portions. The rash consists of small red, scattered flat papules, circular or ovoid in shape, which rapidly develop into vesicles. They usually come out in irregular crops, fresh spots continuing to appear among the older, so that by the fourth or fifth day they are seen in all stages of evolution and involution. Some few of the papules do not develop into vesicles at all but undergo complete resolution in the course of several hours. Nearly all of them develop into vesicles which are fully formed within twenty-four hours. Not infrequently the papular stage is so brief that the fully developed vesicle appears upon skin that shortly before seemed entirely normal. The vesicles are usually at first hemispherical and appear to be superficially situated in the skin. Their contents are limpid, so that they sometimes present the appearance of a drop of clear or faintly yellowish fluid resting upon the surface. In the course of a few hours they become milky and then seropurulent and in a further brief period desiccation takes place with the formation of flat, yellowish-brown, firmly adherent crusts, which separate in about a week, leaving in the majority of instances no scar. The vesicles are readily ruptured by scratching and other injury. Under these circumstances, and when the lesion approaches more nearly to that of variola and involves the deeper structures of the skin, pitting may result, especially upon the face. It is probable that spontaneous rupture of the vesicles does not occur. The pock in varicella commonly has little or no areola, but in the severer cases marked infiltration and hyperæmia of the surrounding skin may be observed. The diameter of the vesicle varies from 1 to 15 or 20 millimetres; their number from ten to hundreds. They are in most cases discrete, but when very numerous confluence may often be discovered upon careful search. Primary umbilication does not occur, but as desiccation takes place a depression in the centre of the crust—secondary umbilication—is sometimes seen. The eruption occurs upon the mucous membrane of the mouth. It is rare upon the conjunctivæ

and upon the labia and prepuce. In these situations, under the influence of warmth and moisture the roof of the vesicle is rapidly destroyed and the lesion converted into a circumscribed superficial ulcer. The peripheral lymph-glands are not infrequently slightly swollen and tender. In the lighter forms there is little or no elevation of temperature during the whole course of the disease; in the more severe cases fever, if not previously present, develops with the rash, to the abundance of which it bears, however, no constant relation. It commonly subsides in two or three days and very seldom lasts a week. It does not conform to type.

The duration of the attack is variable. Three or four weeks may elapse before the separation of the last crusts. Relapses do not occur. The immunity acquired is in most instances permanent. In rare cases subsequent attacks have been observed.

The anomalies of the disease relate to the rash. In rare instances some of the vesicles contain blood, with ecchymoses and bleeding from the mucous surfaces—*Varicella hæmorrhagica;* still more rarely they develop into bullæ like those of pemphigus or ecthyma—*V. bullosa;* in cachectic children some of the skin lesions may become extensively ulcerated or even gangrenous, and death occur as the result of exhaustion—*V. gangrenosa vel escharotica.*

Diagnosis.—Direct Diagnosis.—Varicella in cases seen from the beginning is easily recognized. The mildness of the initial symptoms, the persistence of fever if present upon the appearance of the eruption, the character of the individual pock, which is essentially vesicular, its rapid evolution, the absence of primary umbilication, the appearance of the lesions in irregular crops, so that papules, vesicles, and crusts are seen at the same time in the same region, are of diagnostic importance. Varicella is usually endemic in cities; variola occasionally epidemic.

Differential Diagnosis.—This important matter principally relates to the discrimination of varicella from smallpox, and the chief points are indicated in the foregoing paragraph. In smallpox the onset is abrupt; the fever high; headache and backache intense. There are cases in which, at the period of desiccation, the differential diagnosis cannot be made.

Prognosis.—Varicella is a benign affection. Convalescence is in the majority of cases uneventful and complete. In rare instances death has resulted from nephritis, sepsis, or laryngitis.

VI. SCARLET FEVER.

Scarlatina.

Definition.—An acute, infectious febrile disease, occurring sporadically and in circumscribed epidemics, and characterized by erythematous angina, a diffuse uniform exanthem followed by desquamation, and a tendency to glomerulonephritis.

Etiology.—Predisposing Influences.—No region can claim immunity from scarlet fever. It occurs in every climate and attacks all races. It may prevail at any season, but, owing to the mode of life and the closer

intercourse of school children in autumn and winter, epidemics are more extensive and severe at these seasons of the year. Among personal conditions predisposing to this disease age plays an important part. Children at the breast are rarely attacked. More than half the cases occur before the fifth year and 90 per cent. before the tenth. Adults occasionally contract the disease. After puberty the liability rapidly diminishes. Sex is without influence.

Exciting Cause.—The specific pathogenic germ is not yet known. Streptococci have been demonstrated in the skin and the blood during life and in the viscera after death, and the disease has been regarded as a streptococcus infection. These micro-organisms are, however, present under widely different conditions. Mallory found between the epithelial cells of the epidermis a protozoon which assumed rosette forms like the malarial parasite. Inoculation experiments upon human beings have demonstrated the presence of the infecting agent in the blood, the tears, the secretions of the nose, larynx, and bronchi, the urine, the desquamating skin, and in the contents of miliary vesicles. It is probably eliminated in the discharges from the bowels. The disease caused by artificial inoculation is usually of severe form. The infecting principle is virulent and tenacious. It is transmitted directly from the sick to the well and indirectly by means of fomites. These may be the clothing of the patient, the bedding and furniture of his room, toys, books, letters or other articles with which he has been in contact or which have been exposed to an atmosphere bearing the fine dust made up of his dried secretions or the particles of his desquamating skin. The vessels he has used and remnants of food may also convey the disease. Persons, themselves insusceptible, may be the carriers of the contagion to others at a distance. Physicians and nurses are especially exposed to this risk and the experience of the medical profession in this respect is peculiarly sad. The only way to escape catastrophe is to realize its danger. Household pets, as cats, dogs, and birds, may under certain circumstances carry the disease. Several epidemics have been traced to milk supply. It is probable, as shown by Dornblüth, that the milk has been merely the means of conveyance. The cream is especially liable to cause the disease, and the risk is avoided by boiling. The poison is not borne to any great distance in the air. Its virulence is greatly diminished by oxygen, sunlight, and diffusion. On the other hand, it shows under opposite conditions a vital persistence greater than that of any other disease. Articles of clothing folded and packed away have given rise to the disease after months and even after years. The time at which the patient becomes a source of contagion has been the subject of controversy. It is not only probable but it is also safe to assume that transmission may occur at any time, from the beginning of the attack until desquamation is completed, or, in the case of a pathological discharge, as in otitis media, for an indefinite period. The infecting principle gains access by way of the inspired air. Occasionally the attack of scarlet fever is complicated by another specific infection, as erysipelas, measles, pertussis, varicella, enteric fever, or diphtheria. The Klebs-Löffler bacillus may, however, be absent in throat affections of the most severe character, even those in which pseudomembrane is conspicuous. It is now thought that

the febrile and desquamative diseases to which certain of the domestic animals, as the cat, dog, and horse, are subject—forms of "distemper"—are not, as was formerly supposed, identical with scarlet fever in the human being.

The individual predisposition to scarlet fever is much less general than to measles, variola, and many other contagious diseases. Many persons upon exposure escape. Individuals sometimes escape upon close exposure and contract the disease years later. Certain families show an immunity transmitted from generation to generation; on the other hand many families suffer from a fatal liability. The immunity after puberty is not wholly due to previous attacks. The attack confers an immunity which commonly is permanent. Second attacks are very rare. I have seen three attacks separated by intervals of several years in the same person. Certain individuals who possess an immunity acquired by the attack in early life suffer from slight sore throat when exposed to the infection.

The disease frequently occurs sporadically under circumstances in which it is impossible to trace its transmission. Any such case may become the focus of an epidemic. General epidemics are made up of series of local or circumscribed outbreaks. They last longer than epidemics of measles, sometimes several months, and show remarkable remissions and exacerbations. Epidemics of scarlet fever differ greatly in the severity of the primary disease and in the prominence of severe complications.

Symptoms.—The period of incubation varies from one to seven days; in the majority of cases the disease develops on the third or fourth day after exposure. A short incubation is commonly followed by a severe attack.

(a) Stage of Invasion.—The onset is usually abrupt, without prodromes. It very often comes on at night. The intensity of the initial symptoms is frequently in direct relation to the severity of the subsequent course of the disease. A chill is rare; convulsions are common, especially in young children. Vomiting may be the first symptom. It is in many cases repeated. The temperature rises in the course of a few hours to 104°–106° F. (40°–41.1° C.), and the skin is extremely dry and hot. In very mild cases the temperature may not exceed 101°–102° F. (38.3°–38.9° C.). Older children complain of sore throat and upon inspection the mucous membrane of the soft palate, tonsils, and pharynx shows a uniform erythematous blush varying in intensity according to the severity of the case. The hard palate shows a finely stippled punctiform rash. Febrile phenomena are marked—a furred tongue, red at the tip and edges, refusal of food, thirst, scanty, high-colored urine, restlessness, somnolence, and delirium. Cough and other symptoms of implication of the respiratory tract are slight or altogether absent.

(b) Stage of Eruption.—The eruption appears during the latter part of the first or in the course of the second day, coming out first upon the neck, chest, especially in the infra-axillary regions, and spreading over the face and body to the extremities with such swiftness that in the course of from twenty-four to thirty-six hours the entire surface is covered. It consists at first of minute, close-set red points, coarser and more widely separated upon the legs than elsewhere. The intervening skin rapidly becomes reddened and slightly swollen. In severe cases œdema of the hands and fingers is often marked and a like condition of the eyelids occurs.

The fully developed rash upon the back, abdomen, and thighs is of an intense scarlet or violet-red color, difficult to describe or reproduce and quite unlike that of any other eruptive disease. It has been compared, but very inappropriately, to the color of the boiled lobster. Its distribution over the surface of the body is, in the great majority of cases, nearly uniform, with the common exception of a ring around the mouth which, remaining free, is in striking contrast with the rest of the face. Exceptionally the face and neck only are involved, or the body and extremities, and in some instances, especially in adults, the rash is irregularly patchy in areas varying in diameter from 5 to 20 cm., oval or circular, not abruptly

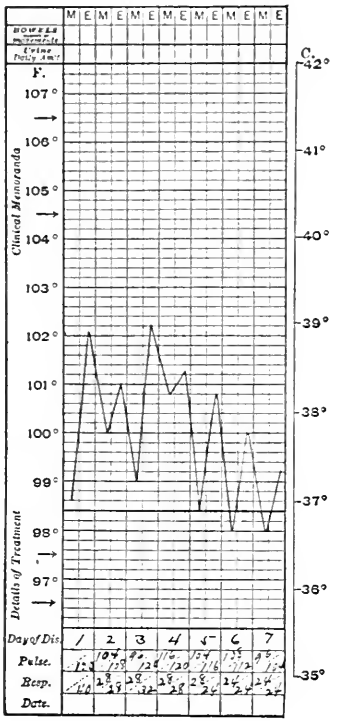


FIG. 228.—Scarlet fever.

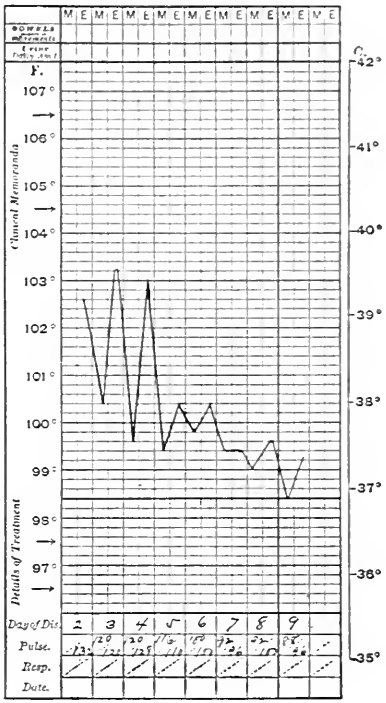


FIG. 229.—Scarlet fever.

marginate but shading off into the surrounding skin. The eruption is largely hyperæmic and the white streak produced by drawing the finger over the surface immediately disappears.

Sudamina may appear and in a group of cases—scarlatina miliaria—the skin is extensively covered with minute yellow vesicles. In the more intense eruptions punctiform hemorrhages may appear and in malignant cases petechiæ and extensive subcutaneous hemorrhagic effusions.

Subjectively sensations of tension, burning, and pricking, together with more or less intense itching, attend the eruption and to these symptoms the restlessness and jactitation in many of the cases are largely due. Swelling of the peripheral lymph-nodes, especially in the region about the angles of the jaw, is common and in some instances they are very tender

The rash reaches its height upon the third or fourth day and gradually fades first where it first appeared. By the end of the first week it has in most instances wholly vanished.

The tongue is at first covered with a thick whitish fur through which project the tips of the swollen red papillæ—an appearance described as the strawberry tongue. In the course of a few days the coating separates, leaving the whole dorsum of the tongue red and rough, the so-called strawberry or raspberry tongue of writers. These terms are neither exact nor descriptive and ought to be abandoned. The bowels are, as a rule, constipated. Diarrhœa may be an early symptom and is sometimes severe. It may occur as the result of a simple catarrhal enteritis or of a dysenteric colitis with tenesmus and bloody stools. In grave cases meteorism may be present.

In a majority of the cases there is simply an erythematous angina with slight enlargement of the tonsils and palatine arches; in other cases these and the contiguous structures are intensely swollen and infiltrated and foci of suppuration may appear; finally, more or less extensive pseudomembrane may develop with intense inflammation, cervical adenitis, and inflammatory infiltration and œdema of the tissues of the neck. The ordinary inflammation is due to the scarlatina poison; the graver forms and many of the pseudomembranous inflammations to secondary infection by pus-producing micro-organisms, while true diphtheria occurs as a complication resulting from infection by the Klebs-Löffler bacillus. Membranous laryngitis is rare in scarlet fever and still more rare is the development of a membranous (croupous) exudate in the bronchi.

The temperature conforms much less closely to type than that of measles or variola. It is much modified by secondary infections. Its rise is abrupt, to 104°–106° F. (40°–41.1° C.); it remains with slight morning remissions about this level until the eruption is fully developed, about the third day. From this point in uncomplicated cases the temperature gradually falls coincidently with the fading of the rash, until about the end of the first week the defervescence is complete. In mild cases the maximum temperature may not exceed 102° F. (38.9° C.), while in the gravest cases hyperpyrexia may occur—105.8°–109° F. (41°–42.8° C.). The temperature often reaches subnormal ranges and the morning remissions may continue to be subnormal for several days.

The pulse is rapid, 120–160; its tension notably increased. In uncomplicated cases the erythrocytes and hæmoglobin are but moderately decreased. Polymorphonuclear leucocytosis is marked from the onset—30,000, even as high as 60.000, and the eosinophiles are increased. In postscarlatinal nephritis profound anæmia rapidly develops. The area of splenic dulness is increased and the lower margin of the organ may be palpable. The area of liver dulness is not enlarged. The urine during the period of invasion and in the early days of the eruption may show a trace of albumin with a few tube-casts—toxic or febrile albuminuria. This disappears with the defervescence. Systematic examination of the urine should be made at intervals of two or three days until desquamation has ceased and convalescence is fully established.

(c) Stage of Desquamation.—Desquamation follows the disappearance of the rash. It may begin at once or not until after several days have elapsed. It first shows itself, as a rule, upon the neck or chest; sometimes upon the abdomen above the inguinal folds. It bears some relation to the intensity of the eruption, being in mild cases furfuraceous, in grave cases lamellar or membranous. The thicker layers of the skin upon the hands and feet may come off in extensive patches and children sometimes remove the epidermis of several fingers like the rag of a glove. The average duration of the desquamation is about two weeks, but the process is in many cases repeated and may continue for five or six weeks. The palmar and plantar surfaces are the last to cease shedding. Occasionally the hair falls out.

Fig. 230.—Desquamation upon face, neck, and chest after scarlet fever.—After Welch and Schamberg.

Varieties.—The cases differ greatly in severity and duration and may be arranged in the following groups:

1. The Ordinary Form—Scarlatina Simplex.—This variety has been outlined in the foregoing general description. It presents varying grades of severity, but the symptom-complex is well defined and the acute process in uncomplicated cases comes to an end by the sixth or seventh day.

2. Larval or Undeveloped Forms.—(a) *Scarlatina Afebrilis.*—The objective phenomena of illness are absent and the patient does not regard himself as sick. Fever is absent or slight—100° F. (37.8° C.), and lasts only a few hours. The rash also is faint, usually limited to the throat and chest, and transient. (b) *Scarlatina sine Eruptione.*—There may be sudden fever with sore throat and vomiting but the skin remains free from eruption. At most there is a transient faint erythematous blush such as is often observed in acute indifferent febrile attacks in young persons of blond complexion. (c) *Scarlatina sine Angina.*—Fever may be present and the eruption may be more or less well defined but the appearance of the throat remains normal throughout the sickness.

These incompletely developed cases often give rise to great difficulties in diagnosis. They occur with some frequency in certain epidemics and are to be recognized by the epidemic tendency, the severe complications which frequently develop, the occurrence of postscarlatinal nephritis, and the fact that they may become centres of infection. The afebrile forms

are especially likely to spread the disease. It not infrequently happens that, in a family of children, one may apparently escape the attack and play about as usual, until, as the others recover, desquamation or sudden pallor, dropsy, and albuminuria make it evident that he also has suffered the infection but without the usual symptoms.

3. Malignant Forms—Scarlatina Maligna.—(a) *Scarlatina Siderans.*—These cases occur sporadically and during severe epidemics. The child is overwhelmed at once by the intensity of the infection. Blinding headache, vomiting, convulsions, hyperpyrexia, delirium passing rapidly into coma succeed one another in appalling succession. There is suppression of urine; the heart's action is progressively rapid and feeble and dyspnœa occurs. Death ensues within twenty-four or thirty-six hours. (b) *Scarlatina Hæmorrhagica.*—Petechiæ appear and develop in a brief time into vibices and extensive suggillations. Epistaxis and hæmaturia are common. All the evidences of a profound toxæmia occur and death takes place in the course of two or three days. Enfeebled and badly nourished infants and especially the inmates of public institutions are particularly liable to this form of scarlet fever, but it occasionally occurs in well-nourished children living under favorable circumstances.

4. The Anginose Form—Scarlatina Anginosa.—The throat symptoms early dominate the clinical picture. Intense inflammation of the tonsils and contiguous structures is attended with pain, dysphagia, and mechanical dyspnœa. Pseudomembrane develops and may extend over the soft palate and uvula into the nasopharynx and nasal chambers or downward to the larynx. The Eustachian tube is involved with infection of the middle ear. Local necrosis takes place and the fetor is almost unbearable. The glands of the neck are enlarged. General sepsis frequently results with fatal termination. If life be prolonged the separation of the sloughs may give rise to fatal hemorrhage from the carotid artery.

5. Protracted Form—Scarlatina Typhosa.—Severe and prolonged cases with delirium, profound prostration, sustained high temperature, and all the evidences of grave general infection. Gastro-intestinal disturbances and marked tympany may be present. The fever may continue for two or three weeks and terminate in lysis. This form has been described also as septic or adynamic scarlatina.

6. Surgical and Puerperal Scarlatina.—Some years ago the subject of scarlet fever after surgical operations and in the lying-in woman attracted much attention. Scarlatiniform rashes may occur under two conditions: First, the patient may, at the time of operation or confinement or thereafter, contract scarlet fever and present all the symptoms of the disease; in this case the surgeon or accoucheur must question himself as to his part in conveying the infecting principle; and second, the patient may develop an erythema as the result of sepsis or as a drug exanthem. The angina and peculiar appearance of the tongue are not present. The distribution of the rash is irregular and its course not that of the eruption of scarlet fever. Local erythema may result from carbolic acid or sublimate solutions of undue strength, and general rashes from certain drugs, as copaiba, oil of santol, and quinine. Desquamation may occur as the result of intense erythema due to any cause. These cases have become infrequent under the methods of modern surgery.

Complications and Sequels.—The complications of scarlet fever are numerous. They are mostly due to secondary streptococcus infection, the primary infection rendering the tissues vulnerable and lowering the powers of resistance of the organism. They are usually severe and have much to do with the gravity of the disease. Some epidemics are characterized by the prominence of certain complications. Almost any tissue of the body may become the seat of inflammation and abscess formation during an attack. The following are more commonly involved:

(a) Throat.—Pseudomembranous and necrotic inflammation of the faucial structures occurs in the anginose form. It is described as scarlatinal diphtheria and may develop during the stage of invasion or upon the appearance of the exanthem. In grave cases there is extensive inflammatory œdema of the neck—"collar of brawn." General septicæmia is apt to supervene and in many epidemics the throat affection is the cause of high mortality. As a rule the throat affection is due to streptococcus infection; much less commonly there is a true complicating diphtheria.

(b) Kidneys.—Nephritis constitutes the most common and important complication. Toxic or febrile albuminuria occurring at the height of the fever is usually transient and without greater significance than the same condition in other diseases, as pneumonia. Much more important is the albuminuria which comes on with the fading of the rash or during the stage of desquamation. The anatomical condition in the quickly fatal cases is that of a glomerulonephritis of varying intensity. Clinically the following grades may be recognized:

1. *Mild Catarrhal Nephritis.*—The urine remains normal in amount. It contains a moderate amount of albumin and a few tube-casts. Cylindroids are present. There is no blood. Œdema is slight and is confined to the eyelids and the pretibial areas. The process may last a few weeks and terminate in complete recovery or it may be the starting point of a chronic nephritis. Sudden intensification of the symptoms may occur with anuria and fatal uræmia, œdema, or effusion into the serous sacs or the larynx.

2. *Graver cases* with increased albumin, casts, and blood. The urine is diminished and there is slight or moderate œdema of the face and lower extremities. Effusion into the serous sacs may occur. The symptoms are not urgent, but anæmia is pronounced and the nephritis tends to become chronic. Uræmia may suddenly develop. When apparent recovery takes place renal inadequacy is often established, and the albuminuria of adolescence or the nephritis following an acute illness or exposure to cold years afterwards may be traced to this condition. These are the cases that under well-directed treatment frequently end in recovery.

3. *Very severe cases* with rapidly developing intense anæmia, general anasarca, anuria, or the passage of small amounts of bloody or porter-colored urine which coagulates upon the application of heat and is loaded with blood and casts. Uræmic accidents, vomiting, facial spasm, convulsions, and coma promptly occur, and despite treatment a large proportion of the cases die.

In rare instances œdema may occur without albuminuria. Under these circumstances the dropsy may change its location, coming and going

without apparent cause. It may be associated with effusion into the serous sacs, especially ascites, œdema of the glottis or sudden pulmonary œdema. This form of œdema may be the result of anæmia or cachexia.

(c) Joints.—Rarely during the acute attack, more commonly in the second or third week, there may develop inflammation of the joints, synovitis scarlatinosa, so-called scarlatinal rheumatism. The cases may be arranged in two groups: 1. Those in which a serous synovitis of more or less intense character is present, involving most frequently the small joints of the fingers, the wrists and knees, exceptionally the spinal articulations, and terminating in complete resolution in the course of a few days. Several joints are usually affected—polyarthritis—but there is little or no rise of temperature and the joint affection lacks the fugacious character so marked in rheumatic fever. This form has been regarded as due to the scarlatinal poison. 2. Suppurative arthritis, usually implicating a single joint and appearing as a local manifestation of a general septic process. Streptococci have been demonstrated in the intra-articular exudate.

(d) Heart.—Benign endocarditis may occur alike when joint complications are present and in their absence. Malignant endocarditis is very rare. Pericarditis is much less frequent than endocarditis. If effusion takes place it may be purulent or, in grave cases, hemorrhagic. Myocarditis is not uncommon. Hypertrophy and dilatation are constant accompaniments of scarlatinal nephritis in children; they are less frequent in adults. It is of importance to perform auscultation of the heart and lungs as a matter of daily routine in every case of scarlet fever. There is no reason why rheumatic fever may not occur as a complication of scarlatina and account for the joint affection and cardiac lesions in certain cases.

(e) Respiratory Organs.—Bronchitis and inhalation pneumonia—bronchopneumonia—are present in cases attended with severe lesions of the upper air-passages. Hypostatic congestion is common in the gravest cases. Croupous pneumonia is rare. Pleurisy is relatively common. It may be plastic; more frequently it is serofibrinous or purulent; rarely hemorrhagic. It usually develops about the middle of the second week.

(f) Auditory Apparatus.—Infection of the middle ear by way of the Eustachian tube is very common. It occurs in almost every case of anginose scarlatina and is attended by serious dangers, both near and remote. In about 85 per cent. of the cases both ears are affected. Suppurative otitis media results with perforation of the tympanic membrane. The inflammation may extend to the labyrinth or to the mastoid cells. Extensive necrosis of parts of the temporal bone may result. Paralysis of the facial nerve is a rare sequel. Meningitis, brain abscess, and sinus thrombosis may occur. In the absence of those accidents spontaneous healing may take place in the course of a few weeks. More commonly the otitis becomes chronic and in many cases, despite treatment, gives rise to permanent impairment or loss of hearing. Burkhardt-Merian found among 4309 cases of acquired deaf-mutism 445, or 10.3 per cent., to be due to scarlet fever.

(g) Lymphatic Glands.—Implication of the lymphatic system occurs in all cases. In mild scarlet fever there is commonly some degree of enlargement of the superficial lymph-nodes, especially in the neck. It is, however, slight and undergoes resolution during convalescence. In more serious

cases the glands may be enormously swollen with cellulitis of the surrounding tissues. Phlegmonous inflammation—angina Ludovici—may occur and lead to extensive necrosis with erosion of vessels and fatal hemorrhage. Retropharyngeal abscess is a very rare complication. The enlargement of the cervical glands is occasionally persistent.

(h) Among the RARER COMPLICATIONS and sequels are enterocolitis, local periostitis, noma, perforation of the soft palate, symmetrical gangrene, and various palsies. Protracted anæmia may occur and the growth and development of the child may be greatly retarded. Acute psychoses, mania, or melancholia sometimes develop during convalescence. These conditions are commonly of brief duration, coming to an end in the course of some hours or days. Exceptionally they last several weeks. Hereditary predisposition is present in many of the cases. Boys more frequently suffer than girls. The prognosis is uniformly favorable.

Diagnosis.—The DIRECT DIAGNOSIS rests upon the sudden onset with rapid rise of temperature, vomiting, and nervous disturbances, as convulsions or stupor, the erythematous angina often accompanied with pseudomembranous exudate, the peculiar tongue, the exanthem, the desquamation, and the swelling of the superficial lymph-nodes. The prevalence of an epidemic or a history of exposure is of importance in doubtful cases. There are cases in which for a time the diagnosis must remain uncertain.

DIFFERENTIAL DIAGNOSIS.—1. MEASLES.—Less abrupt onset, catarrhal symptoms, Koplik's sign, longer period of invasion, a distinctly maculopapular rash, coarse, measly, and thick-set, with an irregularly crescentic arrangement, the less intense sore throat, the absence of leucocytosis, and the fine desquamation are important criteria. The rash appears upon the third or fourth day or later, and first upon the face. It is duller in hue than that of scarlet fever. In rare cases of measles in which a uniform, vivid red rash covers the face and body, maculopapules will be found about the wrists or ankles.

2. RÖTHELN.—The eruption may closely resemble that of scarlet fever. The disease is usually slight; fever and constitutional disturbances are insignificant; angina is absent or trifling; the adenitis involves the postauricular glands to a greater extent than the anterior chains; and the prevalence of an epidemic is of diagnostic importance.

3. SEPTICÆMIA.—Not only so-called surgical and puerperal infection but other forms of septicæmia may present rashes almost identical with that of scarlet fever. Certain of these cases show also erythematous sore throat with a tongue thickly furred and red at the edges and tip, and if death does not occur desquamation may take place precisely as in scarlet fever. The differential diagnosis is difficult and may in any given case be impossible.

4. DIPHTHERIA.—False membrane may develop early and present the appearance of true diphtheria. Scarlet fever with intense pseudomembranous angina, diphtheria with an erythematous rash, and the coexistence of scarlet fever and diphtheria are to be considered. The early membranous sore throat of scarlet fever does not usually show the presence of the Bacillus diphtheriæ, which is, however, usually present in the later forms. The erythema of diphtheria is comparatively rare and when present

lacks the uniform distribution and the intense scarlet color of that of scarlet fever. In every suspected case a bacteriological examination must be forthwith made.

5. Other Acute Infections.—In rare instances rashes suggestive of scarlet fever occur in influenza, cerebrospinal fever, variola,—the initial rashes,—varicella, and enteric fever. The differential diagnosis must rest upon the symptom-complex in individual cases. As a rule little real difficulty arises and even that is dispelled in a brief time.

6. Acute Exfoliative Dermatitis. — Sudden onset with fever, scarlatiniform eruption rapidly becoming universal and fading after five or six days with membranous desquamation present a problem in diagnosis difficult of solution. The absence of angina and the tongue of scarlet fever, the occurrence of alopecia, the loss of the nails, a tendency to recur periodically, the occurrence in adults, and the sporadic character of this rare disease in which the case never becomes a focus of contagion usually render the diagnosis a simple matter. Cases first seen during the stage of erythema sometimes present great difficulties in diagnosis.

7. Erythema Simplex.—This dermatosis occurring in young children may suggest scarlet fever. The trifling constitutional disturbance, the transient eruption, the absence of angina, and in most cases absence of desquamation are of diagnostic value.

8. Drug Exanthems.—Local erythematous rashes may follow the application of mustard, solutions of carbolic acid, corrosive sublimate, or other irritants. Circumscribed or diffuse eruptions may follow the internal administration of belladonna, quinine, the iodides, more rarely chloral, sulphonal, antipyrin, turpentine, and the hypodermic injection of tuberculin. These rashes are not accompanied by fever, a fact which may be rendered unavailable in diagnosis by the presence of fever in the condition for which the drug is administered; nor by angina, save in the cases of belladonna, in which dryness and redness of the throat are conspicuous; and certain of them occasion symptoms which are distinctive—tinnitus in the case of quinine, coryza in iodine, and so forth. The difficulty in diagnosis is usually slight.

It may be said that the greater the experience of the practitioner in the acute exanthemata, the greater his hesitancy to make a positive diagnosis in doubtful cases. The only safe rule of practice is to regard every uncertain case as a possible source of infection and treat it accordingly until the diagnosis becomes clear.

Prognosis.—The mortality varies greatly in different epidemics. It is modified by the severity of the infection and the prevalence of grave complications and is lower in private practice than in hospitals. In some outbreaks the disease has been benign, the death-rate not exceeding 5 per cent.; in others, exceedingly severe, with a maximum mortality of 30 or even of 40 per cent. The prognosis in individual cases is influenced by the following conditions: 1. Age. The danger is much greater in infancy and early childhood than later. A large proportion of the deaths occur between the third and sixth years. The absolute mortality among adults is low but my observation leads me to believe that, in proportion to the number of cases, scarlet fever after puberty is very fatal. 2. Sex. It is said that the

mortality is higher among males than females. 3. The previous health of the patient. Delicate, poorly nourished, and sickly children bear the disease badly. Like the other acute infections scarlet fever is severe and attended with a high death-rate in orphan asylums and similar institutions.

The danger is great in proportion to the intensity of the primary infection as shown by early high fever, stupor, delirium, restlessness, the evidences of general septic infection, and the prominence of local infective processes, such as membranous or suppurative angina, cervical cellulitis, laryngeal obstruction, bronchitis, bronchopneumonia, pleural effusion, heart complications, and nephritis. Hemorrhagic cases are usually fatal. A persistently high pulse-rate is of itself an unfavorable sign.

The prognosis must in all instances be guarded. Apparently benign cases may develop the gravest symptoms or fatal nephritis may occur during convalescence. No case, however favorable, can be regarded as out of danger until at least four weeks have elapsed from the onset of the attack.

VII. MEASLES.

Rubeola; Morbilli.

Definition.—An acute febrile infection occurring in epidemics, and characterized by initial coryza, bronchial catarrh, and a generalized maculopapular eruption.

Etiology.—Predisposing Influences.—The susceptibility to measles appears to be almost universal. The disease prevails in every climate and attacks all races. Negroes suffer more severely than whites and are more liable to develop grave complications and sequels. *Season.*—Outbreaks take place at all times of the year but are more common in the winter and spring than in the warmer seasons. Measles is peculiarly a disease of childhood. Adults not protected by an attack in early life may contract it and frequently manifest the symptoms in an aggravated form. It is more common after puberty than scarlet fever. Congenital cases have been observed; it is rare during the first six months of life. The great majority of cases occur before the tenth year. The *sexes* are alike liable.

Exciting Cause.—The specific pathogenic cause has not been demonstrated. Inoculation experiments upon human beings have shown the presence of the infecting principle in the blood, in the tears, in the secretions of the nasal, pharyngeal, and bronchial mucous membrane, and in the contents of vesicles occasionally present. Inoculation with the epithelial scales thrown off at the close of the disease has been unsuccessful. Transmission of the disease under ordinary circumstances takes place by the breath or the nasal or bronchial secretion—droplet infection. It may result directly from close approach or contact with the patient, or remotely through the conveyance of the poison by a third person or by fomites. Measles is very commonly disseminated in school or upon playgrounds by children who appear to be suffering from ordinary nasal or bronchial catarrh but who are in reality in the pre-eruptive stage. The infecting principle is intensely active but neither tenacious nor persistent as is that of scarlet fever. The

disease is communicable during its whole course from the earliest appearance of coryza. The individual predisposition to measles is so general that upon exposure very few escape. The adult who suffers has, as a rule, not been exposed to the infection in childhood and is often an only child who has been educated at home. A congenital immunity seems to exist in rare instances. In the majority of cases an acquired immunity results from the attack. Second, or even third, attacks may occur at intervals of some years, but they are infrequent. I cannot agree with those who regard multiple recurrences as common, since my experience coincides with that of Jürgensen and Eichhorst in Germany, and Holt in this country, who hold that second attacks are rare. Measles in cities is in a certain sense endemic. Sporadic cases occur at intervals and constitute the starting-point of more or less extensive epidemics. When the susceptible individuals in the affected locality have had the disease the epidemic ceases. The poison is frequently carried to neighboring regions by persons in the period of incubation or in the pre-eruptive stage, who become centres of infection for new local epidemics. Extensive outbreaks occur at intervals of five or six years and at long intervals the disease becomes pandemic. Occasionally other epidemic diseases of children, especially whooping-cough and varicella, precede, accompany, or follow outbreaks of measles. Outbreaks are common in time of war among the younger recruits and conscripts, many of whom come from country districts in which the disease has not prevailed for long periods.

Symptoms.—The period of incubation is from seven to fourteen days, usually about ten. In artificially inoculated cases it is commonly less than ten. Prodromes are common. They consist of loss of appetite, restless sleep, fretfulness, and in many cases feverishness or light fever.

(a) Stage of Invasion.—The prodromal symptoms are intensified. There is chilliness which may be repeated, sometimes shivering, but convulsions and distinct chills are uncommon. The temperature rises, reaching 102°–104° F. (38.9°–40° C.) upon the first or second day. It then falls a degree or more to rise again upon the appearance of the exanthem. Nausea, vomiting, and headache are present. The tongue is furred. Coincidently with the appearance of these symptoms coryza develops and is often intense. The phenomena are those of an ordinary severe influenza. Irritation and smarting of the eyelids, lachrymation, photophobia, persistent sneezing, running at the nose, sore throat, discomfort in swallowing, hoarseness, and cough, at first of a brassy or croupy character, develop one upon the other in rapid succession and varying intensity. These initial catarrhal symptoms are characteristic and occur in the mildest cases in which chilliness, fever, and the associated signs of the reaction of the organism to general infection are not observed. Upon inspection the vessels of the conjunctivæ are injected, the eyelids swollen, the nasal mucosa tumid and reddened. The mucous membrane of the mouth and throat is erythematous, while upon the soft palate and roof of the mouth, and particularly upon the buccal mucous membrane, are to be seen pin-head or split-pea-sized, circumscribed, round or irregularly shaped reddish blotches slightly or scarcely at all raised above the surrounding surface, usually discrete, sometimes confluent. This eruption also shows itself in the larynx and is

doubtless the cause of the laryngobronchial symptoms as it is of the coryza. It has been called the *enanthem* in contradistinction to the rash upon the skin, the *exanthem*. In a strong light there may be seen upon some of the spots upon the mucosa of the cheeks and lips minute bright whitish or bluish-white flecks described by Koplik. These flecks appear early and soon disappear, and since they have not been observed in other diseases are of value in the early diagnosis of measles. The duration of the stage of invasion, or, as it is sometimes called, the catarrhal stage, is commonly three or four days; exceptionally it is shorter or it may be as long as a week.

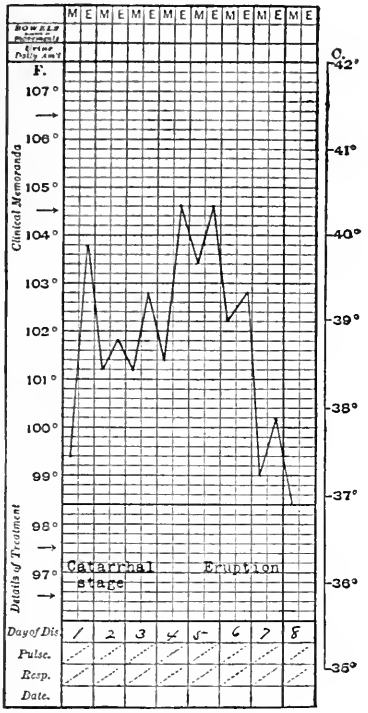


Fig. 231.—Measles.

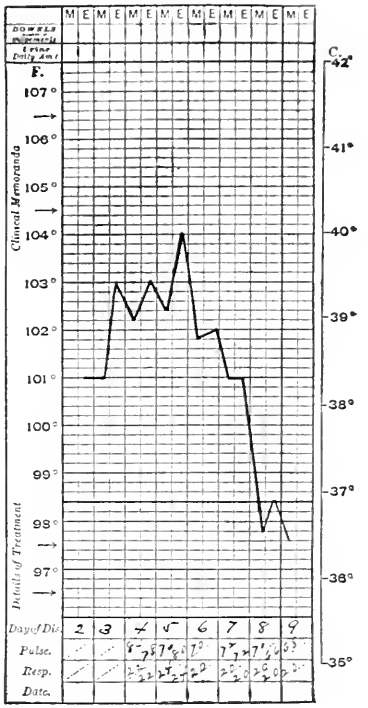


Fig. 232.—Frank uncomplicated measles in a woman aged 52.

(b) Stage of Eruption.—The fever rises and may reach 104°–106° F. (40°–41.1° C.), the pulse-rate may be 140 or higher. Delirium or stupor may be present in the severer cases. The patient complains of heat and burning of the skin, sore throat and general discomfort, and is restless and wakeful. Usually upon the second or third day of the eruption great and rapid amelioration of all these symptoms occurs and the fever which has remained high falls by crisis or by rapid lysis to normal or subnormal ranges. The eruption appears as small red or brownish-red soft flat papules which rapidly increase in size and in numbers. When fully developed the individual spots are irregularly circular or oval and differ greatly in size, the average diameter being that of a split pea. They are unevenly distributed but close set and very often confluent, especially upon the face,

buttocks, hands and feet, where there is also some tumefaction. They are circumscribed and the intervening skin is normal or slightly hyperæmic. Not infrequently a crescentic arrangement may be made out elsewhere. The color momentarily fades upon pressure or upon tension of the skin. The eruption appears first upon the forehead, chin, and cheeks; it invades the hairy scalp and spreads rapidly to the neck, back, hands and arms, anterior surface of the trunk and lower extremities, commonly in the order named, invading the entire surface in the course of twenty-four or thirty-six hours. By the end of the second or during the third day the eruption is fully developed. In severe cases punctiform hemorrhages now appear in some of the papules, especially upon portions of the body exposed to pressure. This condition is not significant and must not be confounded with the true hemorrhagic variety of the disease. From this time the eruption rapidly fades, first where it first appeared, namely, upon the face, and in the course of a further period of two or three days disappears, leaving faint yellowish or dirty-brown areas of pigmentation which in turn gradually pass away. In the beginning of the stage of eruption and in many cases throughout its course the skin is moist and often bathed in free perspiration. At the height of the eruption the superficial lymph-nodes of the neck and elsewhere are often slightly swollen and tender.

(c) Stage of Desquamation.—Following the fading of the rash desquamation takes place in the form of fine branny scales, so fine that the condition may be easily overlooked. This process begins on the face and involves the entire surface, occupying about a week. The catarrhal symptoms in uncomplicated cases gradually disappear, so that, by the end of the second week from the initial coryza, convalescence is fully established. Cough frequently persists. In the absence of inflammatory complications leucocytosis does not occur. Epistaxis is common at the height of the attack; diarrhœa is apt to occur at some time during its course. Relapses of measles are extremely rare.

During epidemics atypical cases occur. They are not common. Variations in the rash may consist: (1) in the development of distinct papules, hard to the touch but not extending deeply into the skin—*morbilli papulosi;* (2) a vesicular form, *m. vesiculari;* (3) cases in which the eruption does not appear, although the general symptoms and the coryza are present—*m. sine exanthema;* (4) cases in which the mucous membranes are not involved—*m. sine enanthema.* To these must be added variations in the constitutional manifestations. In rare cases there is no rise of temperature—*m. afebriles.* In the malignant forms the organism is unable to withstand the intensity of the infection and death takes place in the course of two or three days after sustained hyperpyrexia, profound adynamia, or hemorrhages into the skin and mucous membranes. The malignant forms are very rare in private practice; they occasionally occur in asylums and in the fierce epidemics of camps, and were common in the first outbreak among the natives of the Fiji Islands, where measles prevailed as a veritable scourge. Death may occur before the rash appears, or a few papules may show themselves upon the forehead and wrists. Hemorrhagic or black measles—*m. hæmorrhagica*—is characterized by convulsions, delirium and coma, petechiæ, bleeding from mucous surfaces, and profound constitutional depression.

Complications and Sequels.—Epidemics differ greatly as regards the frequency and severity of complications. In their absence measles is a comparatively benign malady, but they are sufficiently common to place it among the more serious diseases of childhood. Debilitated and badly nourished children living in unhygienic surroundings and those in asylums and institutions are especially liable. The ordinary complications are due to an extension or intensification of the catarrhal processes peculiar to the disease.

Otitis media is not very uncommon. It may result in perforation of the tympanic membrane and permanent impairment of hearing, or lead to sinus thrombosis, meningitis, or abscess of the brain. Purulent conjunctivitis may occur and in neglected cases infiltration and ulceration of the cornea. Catarrhal laryngitis is of frequent occurrence; the pseudomembranous form is rare and very dangerous; œdema of the glottis is very uncommon. Diphtheria is much less frequent in measles than in scarlet fever.

Fig. 233.—Cancrum oris complicating measles.—After Welch and Schamberg.

The catarrhal bronchitis so prominent in the disease is, in itself, without serious significance, and in favorable cases terminates in resolution with the convalescence. Its tendency to extend to the finer tubes and give rise to lobular collapse and bronchopneumonia constitutes the gravest danger. If the involvement of the vesicular structure is limited there is increase of fever with acceleration of the pulse, harassing cough, and disturbance of respiration. The sickness is prolonged but terminates favorably. When the lesions are extensive the symptoms become urgent and a large proportion of the cases die. It is to this complication that the high death-rate of measles under unfavorable circumstances is due. Croupous pneumonia is much less common. Pleural effusion is rare. Acute enterocolitis is a frequent and serious complication in some epidemics. Toxic albuminuria occasionally occurs as in other febrile infections, and the diazo reaction is present. There is little tendency to nephritis. Arthritis is very rare. In young and debilitated children gangrenous stomatitis and in girls gangrene of the pudenda occur during convalescence with greater frequency than in any other infectious disease.

Among the common sequels are chronic local inflammations, conjunctivitis, otitis, nasal catarrh, laryngitis, and bronchitis. The intestinal catarrh arising as a complication may lead to chronic colitis. Tuberculosis is a very common sequel. The lesions of measles are such as to render the patient peculiarly liable to this infection, but the rapidity with which

tuberculous bronchopneumonia and acute miliary tuberculosis develop in many instances renders it probable that a latent tuberculous process has been roused into activity during the attack. Pulmonary tuberculosis is a common remote sequel of measles in the adult, and miliary tuberculosis and tuberculous meningitis may frequently be traced to measles in earlier life. In some cases enlarged caseating bronchial glands may be the starting-point of the general infection. Among the rarer sequels are tuberculosis of the cervical lymph-nodes and of the bones and joints. Palsies occur as the result of peripheral neuritis but are much more rare than in diphtheria or scarlet fever.

Diagnosis.—The DIRECT DIAGNOSIS of well-developed measles after the appearance of the eruption is generally unattended with difficulty. During an epidemic coryza persistent sneezing and fever are suspicious. The appearance of the eruption on the second or third day upon the mucous membrane of the mouth and throat, and Koplik's sign are of positive diagnostic value.

The DIFFERENTIAL DIAGNOSIS concerns: 1. ROTHELN (see p. 75). 2. VARIOLA (see p. 47). 3. TYPHUS FEVER (see p. 40). 4. SCARLET FEVER (see p. 66).

5. SYPHILITIC ROSEOLA usually occurs in the adult. The eruption is polymorphous, the enlargement of the superficial lymphatic glands is greater, and the signs of syphilis are to be found in the mouth and throat and upon the genitalia. The Wassermann reaction may be necessary.

6. DRUG EXANTHEMS.—Exceptionally the administration of salicylates, antipyrin, quinine, turpentine, or copaiba is followed by a rash suggesting rather than resembling that of measles. These rashes are not accompanied by fever or throat symptoms, nor have they the uniform appearance and distribution of the measles exanthem (see p. 683).

In the negro the difficulties in doubtful cases are increased; but the mode of onset, the coryza and bronchitis, and the peculiarities of the rash upon the mucosa of the mouth are of diagnostic importance. The soft flat papules may be distinguished in the darkest skin.

The diagnosis in certain cases must for a time remain doubtful, especially when the disease appears sporadically or prevails during epidemics of rötheln, scarlatina, variola, or typhus.

Prognosis.—The character of the prevailing epidemic and previous condition of the individual greatly modify the prognosis. The death-rate during the first six months of life is relatively low; it reaches its maximum during the second year and rapidly falls after the fifth year. After the twentieth year it rises again. In private practice the mortality is about 3 per cent., in some epidemics not more than 1.8 per cent.; in hospital and asylum practice and in camps and barracks it may reach 30 per cent. The vital statistics of measles are misleading, because the people regard the disease as an insignificant malady and among the lower classes only the more serious cases come under medical observation. Uncomplicated measles is, in point of fact, a benign infection, but the tendency to pulmonary complications renders it one of the gravest diseases of childhood. It is estimated that about one-third of the cases in which bronchopneumonia develops terminate fatally.

VIII. RUBELLA.

Röthein; German Measles; Epidemic Roseola.

Definition. — An acute epidemic infectious disease characterized by a diffuse maculopapular eruption and swelling of the superficial lymphatic glands.

Rubella has some points of resemblance to scarlet fever and to measles and was at one time regarded as a hybrid of the two. It is now known to be an independent substantive affection.

Etiology. — The infecting principle has not yet been discovered. The disease is readily transmissible and usually prevails in extensive epidemics. Outbreaks occur in series, followed by long intervals during which the disease does not recur. In the absence of an acquired immunity persons at any age are susceptible. Rubella does not protect against the infection of scarlet fever or of measles, nor do these diseases protect those who have passed through them against rubella.

Symptoms.—The period of incubation varies from ten to twenty days. Prodromes are usually absent.

Stage of Invasion. — The symptoms are generally mild. They consist of the ordinary manifestations of the reaction of the organism to an infection of little intensity, and the coryza, laryngitis, and pharyngitis which usually precede the exanthemata. The duration of this stage is not constant, varying from a few hours to two or three days. The symptoms may be so slight as to be altogether overlooked and the rash may then be the first indication of illness. The elevation of temperature is trifling, usually about 100° F. (37.8° C.), and rarely exceeding 102° F. (38.9° C.), and transient. In asylums and foundling institutions rubella sometimes prevails as a serious malady.

Stage of Eruption. — The rash commonly appears upon the first day; it may be as late as the third. It shows itself first upon the face and neck, and spreads in the course of twenty-four hours over the body and extremities. It consists of round or oval reddish spots about the diameter of a split pea, mostly discrete, sometimes confluent, and surrounded by areas of hyperæmic skin. In some of the cases extensive tracts of the skin are intensely hyperæmic so that the rash resembles that of scarlet fever rather than measles. The crescentic arrangement of the papules, seen in measles, cannot usually be made out. The eruption frequently fades irregularly in patches some hours after it comes out, so that certain areas of the surface are covered and not the entire body at the same time. In the course of two or three days the rash gradually disappears with fine furfuraceous desquamation, leaving a faint pigmentation which persists for a short time. Slight itching commonly accompanies the rash. The superficial lymphatic glands, especially those of the neck, are slightly enlarged. They undergo resolution in a short time after the fading of the eruption. The suboccipital and lateral chains are commonly involved to a greater extent than the anterolateral. The enlargement of the lymph-nodes in some instances precedes the appearance of the eruption.

Relapses are rare; complications infrequent. Albuminuria, bronchitis, colitis, and pneumonia have been noted. Herpes may occur. There are no special sequels. The immunity acquired by the attack is not always permanent. Second and third attacks may occur.

Diagnosis.—The early cases may present great difficulty in diagnosis. When an epidemic is prevalent the recognition of rubella is easy. The DIRECT DIAGNOSIS rests upon the trifling nature of the disease, the short initial period, the character of the eruption, its patchy distribution, the early enlargement of the glands, and the absence of severe throat symptoms and coryza.

DIFFERENTIAL DIAGNOSIS OF SCARLET FEVER, MEASLES, AND RUBELLA.

	Scarlet Fever.	Measles.	Rubella.
Contagion	Highly contagious	Highly contagious	Variable in epidemics.
Transmissibility	By direct contact, approach, and fomites	By direct contact, fomites, and through the air	Direct contact and fomites; not through the air.
Period of Incubation	Average 2 to 7 days	Average 9 to 14 days	Variable; average 1 to 3 weeks.
Prodromes	Absent or very brief—onset commonly sudden	Commonly 3 to 4 days. Drowsiness and catarrhal symptoms	Slight and of short duration.
Koplik Spots	Not present	Present in about 90 per cent. of cases	Not present.
Vomiting	Common at onset	Infrequent	Rare.
Temperature	High—103°–105°F.—lasting about a week	High, lasting about a week, average 102°–104° F.	Slight elevation, seldom more than 101°–102° F.—subsides in 1 to 3 days.
Catarrhal Symptoms	Commonly absent	Prominent throughout	Slight.
Tongue	Whitish fur, enlarged papillæ; later dry and red	Tongue coated	Slightly coated, not characteristic.
Throat	More or less intense erythematous angina	Moderate redness of mucous membranes	Punctiform red spots over uvula, palate, and pharynx.
Lymph-nodes	Glands at angle of the jaw enlarged	Cervical, postauricular and submaxillary nodes enlarged	General enlargement, especially of postcervical chains.
Pulse	High frequency—120–140	Corresponding to elevations in temperature	Varies with fever.
Urine	Early toxic albuminuria in severe cases. Later signs of nephritis	Albuminuria rare	Albuminuria very rare and slight.
Eruption	First appears on neck and chest, spreads slowly over entire body. Fully developed about the fourth day. Small punctate efflorescence or diffuse blush disappearing on pressure, lasts about a week. Intense scarlet color; usually absent around mouth	First appears on face, spreads gradually over entire body. Fully developed by the second or third day. Consists of small papules arranged in crescentic groups; these are confluent in places; fades in 4 or 5 days; deep red, dusky or purplish	First appears on face, spreading to neck and breast, then to arms, legs and feet. Fades in parts first involved while spreading to others. Two varieties — morbilliform, small, slightly elevated papules, discrete, sometimes confluent; scarlatiniform. Duration 2 to 4 days or less. Color rose red but variable.
Desquamation	Coarse, bran-like; lamellar	Branny	Fine, branny.
Convalescence	Tardy; complications frequent, especially nephritis, otitis media, etc.	Slow; tendency to complications as bronchopneumonia and other infectious diseases, especially tuberculosis	Rapid without complications.

Differential Diagnosis.—Rubella is most frequently mistaken for mild measles or scarlatina. From measles it is distinguished by the want of prominence of catarrhal phenomena, the slighter fever, the brighter hue of the eruption, the absence of the crescentic grouping of the papules, the fact that the adenitis involves to a greater degree the suboccipital and postauricular glands, and the absence of Koplik's sign; from scarlatina by its gradual onset, benign character, the absence of severe throat symptoms, the peculiarities of the rash, the character of the desquamation, the tongue, and the fact that there is no special tendency to nephritis.

Prognosis.—Rubella is a benign disease almost invariably terminating in recovery. In foundling hospitals and asylums it has sometimes assumed unusual severity, and fatal cases have occurred commonly as the result of an intercurrent pneumonia, colitis, or nephritis, rather than of the primary disease (see table on opposite page).

THE FOURTH DISEASE.

In 1900 Dukes described an infectious disease which he called "the fourth disease." This communication attracted considerable attention and the subject has been discussed by a number of clinicians. The incubation period is stated to be about the same as that of German measles, ten to twenty-one days. Prodromes were absent in most of the cases but malaise and a mild erythematous angina were occasionally observed at the time of the appearance of the rash. The evolution of the exanthem was rapid, the entire body being covered in the course of a few hours. Whether or not it was present upon the face is not stated. Its color was like that of scarlet fever but brighter. The superficial lymph-nodes were enlarged. The temperature was subfebrile, not often exceeding 101° F. (38.5° C.). Upon the subsidence of the eruption there was desquamation. Sequels were not observed and the attack did not confer immunity against scarlet fever or rubella. This affection has not been generally recognized as a clinical entity.

IX. WHOOPING-COUGH.

Pertussis; Tussis Convulsiva.

Definition.—An infectious endemic and epidemic disease characterized by hyperæsthesia and catarrh of the respiratory tract and a peculiar, spasmodic cough occurring in paroxysms which terminate in a prolonged inspiration attended by a shrill crowing sound or whoop.

Etiology.—Predisposing Influences.—Whooping-cough is a widely prevalent disease. The individual susceptibility, like that to measles, is almost universal. Very few persons unless rendered immune by a previous attack escape upon exposure. Nearly twice as many cases occur during the winter and spring as during the summer and autumn. It is peculiarly a disease of infancy and early childhood. More than one-half the cases occur during the first two years of life; very few cases after the second dentition. That the immunity after the seventh year is acquired rather than congenital is shown by the fact that in individuals not protected by a previous attack the disease may be contracted upon exposure at any period

of life. Sucklings are not exempt. Sex is without influence in early life, but among adults women are more liable than men, a fact to be explained in part by increased exposure, in part by the more common neurotic constitution in women. Pregnancy appears to be attended with an especial liability. The previous condition of health is of great importance. Delicate children and those suffering from nasal or bronchial catarrh are especially liable to contract the disease upon slight exposure.

Exciting Cause.—Afanassiew, 1887, discovered in the secretions a short bacillus, cultures of which injected into the respiratory passages in animals produce catarrhal inflammation. Koplik more recently found a bacillus resembling that of influenza, which he regards as the cause of the disease. Bordet and Gengou described an organism—Bacillus pertussis—with which the serum from convalescents yields a positive agglutination reaction. The question has not yet been settled.

Clinical experience makes it clear that the infective material is eliminated by way of the mucous discharges and perhaps by the expired air. It reaches the organism with the inspired air. Actual contact, close approach, or fomites, especially such articles as the handkerchief or towel, constitute the usual means of transmission from the sick to the well. Under certain circumstances a third person may readily transmit the infective material. The dried sputum circulating as dust in the atmosphere is probably also a source of infection. The disease is transmissible from the earliest appearance of the catarrhal symptoms, and since it cannot be recognized until the spasmodic stage, and since in suitable weather the patients are kept as much as possible in the open air, the patient alike in the nursery and school and out of doors is in constant danger of disseminating it. The attack confers an immunity which in most cases is lifelong. The occasional occurrence of cases in elderly persons, who have passed through an attack in childhood, living in the house with children suffering from the disease, shows, however, that the protection is not always permanent.

Whooping-cough is endemic in large cities when it takes the form of extended epidemics at irregular intervals of from two to four years. Its prevalence is sometimes so wide-spread as to merit the descriptive term pandemic. Outbreaks are not infrequently associated with epidemics of measles, scarlet fever, or varicella, and these diseases occasionally run their course coincidently with whooping-cough in the same individual.

Symptoms.—The period of incubation varies from seven to ten days. If, after exposure, two full weeks elapse without the development of catarrhal symptoms, the probability becomes very strong that infection has not taken place.

The Course of the Attack.—(a) The *catarrhal stage* begins with the symptoms of an ordinary subacute bronchitis, which gradually increase in intensity. In the course of some days the cough tends to become paroxysmal, the spells being more frequent and severe during the night and after meals. Running at the nose, hoarseness, and a croupy ringing cough, the indications of a nasal and laryngeal catarrh of moderate severity, are associated symptoms. There is very often fever of moderate grade. The duration of this stage is about a week or ten days. Cases vary greatly in

this respect, however, some children developing the whoop in the course of a day or two from the beginning of the catarrhal symptoms, others not until three or four weeks have elapsed.

(b) *Spasmodic or Paroxysmal Stage.*—The fever subsides. The catarrhal symptoms continue and may be intensified. The cough becomes distinctly paroxysmal and characteristic, the attacks ending in a long-drawn "whoop" from which the disease receives its name. The true nature of the disease is now first apparent. The patient experiences a sensation of tickling in the larynx or under the sternum. Little children run terrified to the nurse or mother and cling to her; older persons grasp some object, as the arms of a chair, for support. The fully developed paroxysm usually begins with a long-drawn inspiration which is immediately followed by a series of ten or fifteen short explosive coughs of increasing intensity and repeated so rapidly that breathing is ineffectual until at length a prolonged deep inspiration occurs, during which the whoop is produced. One or more new series of coughs terminating in the whoop may forthwith follow and the paroxysms may not come to an end until a mass of tough stringy mucus is raised. This is usually small but in little children it is often very abundant and must be removed from the mouth by the finger. It may be expelled in the act of vomiting. An abundance of thick mucus is at the same time discharged from the nose. The signs of mechanical disturbance of the venous circulation are conspicuous. The face and neck become congested and cyanotic, the veins of the face and the jugulars stand out prominently, there is protrusion of the eyeballs, sometimes marked injection of the conjunctivæ, and the lips are swollen and blue. As the attack comes to an end the face or the whole body may break out into a more or less profuse sweat. In severe paroxysms the sphincters may be relaxed. Headache and vertigo are common, and at the close of a severe attack the child sinks exhausted upon the mother's lap. The condition is most distressing, but in a little while the child usually recovers himself and goes about his play until another spell occurs. The single paroxysm lasts from fifteen to forty-five seconds, rarely longer; when two or more immediately succeed each other, the whole attack may be prolonged to two or three minutes. They are fortunately not all of the same intensity, frequent milder attacks occurring between those which are more severe. They may be brought on by taking food or drink, especially anything cold, by laughter or vexation, and in some cases by traction of or pressure upon the tongue. They are more frequent in a close room than in the open air and by night than during the day. The number of paroxysms varies from three or four to sixty to one hundred during twenty-four hours. Severe paroxysms after the taking of food almost always cause vomiting and the patients very often become much emaciated and reduced in strength. In many cases, however, as soon as the distressing symptoms are over the child will eat another meal, which should always, under these circumstances, be offered to it. The violence of the cough forces the tongue against the lower incisor teeth and very often causes laceration of the mucous membrane of the frænum, which is followed by superficial ulceration. A marked leucocytosis occurs. In very mild cases the paroxysms are not only less frequent but they are also less violent, and occasional cases occur in which

the whoop is absent throughout the attack, the nature of which is apparent from the presence of other cases in the house, the spasmodic spells of coughing attended with vomiting and terminating with the expulsion of a mass of tenacious mucus, and the protracted course of the illness.

The cough is the result of an extended irritation involving the upper air-passages. It is probable that the gradual accumulation of mucus in the region of the bifurcation of the trachea plays an important rôle in its production. The mechanism of the whoop consists in the forcible indrawing of air through the spasmodically narrowed glottis. The disease has the characteristics of a neurosis affecting the respiratory tract.

Laryngoscopic examination frequently shows the mucous membrane of the larynx to be congested and swollen, especially in the interarytenoid space, and sometimes the seat of hemorrhagic patches or superficial erosions. Irritation of the mucous membrane between the arytenoids or of the posterior surface of the epiglottis with a sound, always causes the paroxysm. A similar condition of congestion and swelling has been observed in the trachea, in which a plug of mucus has been seen just before the paroxysm—Roosbach. The difficulty of such examinations is obvious.

Physical examination yields unimportant signs. The resonance is impaired during the paroxysm and increased at its close. Auscultation yields commonly an enfeebled vesicular murmur and bronchial râles, usually dry—sonorous or sibilant.

The average duration of the spasmodic stage is about one month. The symptoms progressively increase in intensity for a fortnight or longer, remain stationary for a time, and gradually subside. In mild cases this stage may not exceed a week or ten days, or the whoop may be wholly absent; in severe cases it may be prolonged, especially if the patient must be housed, as in the winter, for three or four months, with remissions and exacerbations.

(c) *Stage of Decline.*—The paroxysms diminish in severity and frequency; the expectoration becomes more abundant and less tenacious; and finally, as at the beginning, the symptoms are those of an ordinary catarrhal bronchitis, which varies in intensity and continues two or three weeks in favorable and much longer in unfavorable cases, especially during the winter months when convalescents must be kept housed.

The duration of the attack varies between two and four months. A majority of the cases in older children can scarcely be regarded as ill. They are out of bed and eat well, and in proper weather can pass some hours in the open air. They, however, lose flesh and become pale.

A cough habit is often developed during the attack, and for several months after full convalescence has been established, with every cold or nasobronchial catarrh, however trifling, a paroxysmal cough with whooping returns, and this is particularly the case with the children of neurotic parents. Under these circumstances the disease is not communicated. Relapses practically do not occur. Second attacks are by no means uncommon, but as has already been mentioned they usually occur after the lapse of years, and children and their parents or grandparents often suffer at the same time.

Complications and sequels are numerous and may be arranged in two categories, the mechanical and the infectious.

(a) The *mechanical complications and sequels* are caused by increased respiratory pressure during the paroxysm, or circulatory disturbances. Acute emphysema is common. It is as a rule transient. If it persists pseudohypertrophic emphysema results. Rupture of the tissue of the lung may give rise to interstitial emphysema, or the air may find its way along the peribronchial connective tissue to the anterior mediastinum or upwards and give rise to subcutaneous emphysema of the neck. Pneumothorax is less common. Dilatation of the right heart may occur in consequence of the interference with the pulmonary circulation during the paroxysms. It is possible that valvular disease may, in some instances, be due to the heart strain of severe whooping-cough. The pulse after the paroxysm is often feeble and irregular, and progressively so as the attack goes on. The vomiting is largely due to mechanical disturbance caused by cough. Sometimes the patient vomits freely during several paroxysms daily for periods of weeks, and as a result is greatly reduced in flesh and strength. Partly as the result of the violent succussion and partly from exhaustion, involuntary discharges of gas or fecal matter are of common occurrence in severe paroxysms. Prolapse of the bowel and hernia are common and must be ascribed to the same causes. Involuntary discharge of urine likewise occurs. Pregnant women frequently abort. Very common are lesions of the blood-vessels during the paroxysms, resulting in hemorrhages into the skin, particularly about the forehead and eyes, and into the mucous membranes, especially subconjunctival ecchymoses. Much less common are slight hemorrhages—not more than a few drops—from the ear in consequence of superficial lacerations of the tympanitic membrane. Epistaxis is very frequent, hæmoptysis rare. Hemorrhage from the bowel is very unusual, and when it occurs is due to the mechanical derangements which cause prolapse. Convulsions are not uncommon, especially in very young children, and have been ascribed to the engorgement of the cerebral vessels. Meningeal and cerebral hemorrhages occur, but these accidents are extremely infrequent. Hemiplegia and aphasia may result. Sudden death has occurred.

(b) The *infectious complications* include inflammatory enlargement of the bronchial glands, sufficient in some instances to give rise to dulness over the manubrium; bronchopneumonia, which is very common and the cause of death in the majority of the fatal cases; tuberculosis, which may take the form of a tuberculous bronchopneumonia, miliary tuberculosis, or an acute caseous consumption; croupous pneumonia, which is infrequent; pleurisy, still more rare; and nephritis, likewise very uncommon. Other inflammatory complications are seldom encountered. Transient albuminuria is not infrequent and glycosuria is occasionally observed. Many of the complications are essentially chronic and persist as sequels. The patients not infrequently show an especial predisposition to recurrences of bronchial catarrh. Emphysema and asthma are common sequels.

Diagnosis.—The DIRECT DIAGNOSIS of whooping-cough during the early part of the catarrhal stage is impracticable. In the course of a week the increasing severity of the symptoms and the tendency of the cough to become paroxysmal and worse at night, to cause vomiting, suffusion of the eyes, and flushing of the face, render the diagnosis during an epidemic,

or with a history of exposure, a probable one. A like uncertainty arises in regard to very mild cases. A child may cough for several weeks without having a well-developed paroxysm. If there be no fever, only a few râles now and then upon auscultation, and ordinary treatment be without effect, the diagnosis by exclusion may be made. The occurrence of the whoop renders the diagnosis easy and certain. It is to be remembered that pressure upon or traction of the tongue, the act of swallowing, and emotional disturbances may cause a paroxysm—facts which the physician may use for diagnostic purposes. The diagnosis may also be difficult in early infancy, when the cough attending ordinary bronchitis sometimes assumes a paroxysmal character and is attended with a croupy or crowing sound that is suggestive of the whoop. The ulcer upon the frænum and subconjunctival or other hemorrhages are not apt to occur in mild cases and these only present diagnostic uncertainties.

Prognosis.—Uncomplicated whooping-cough tends to run a favorable course. The great tendency to complications places it, however, among the most serious of the diseases of childhood. It has been estimated that fully two-thirds of the deaths from this disease occur within the first year. After the fourth year the danger rapidly diminishes. Bronchopneumonia and enterocolitis are the most common causes of death. Convulsions very often occur in fatal cases in early infancy. Delicate and badly nourished children, those living under improper hygienic conditions, those who have rickets or who have been debilitated by a recent attack of measles, influenza, or other serious infection are apt to suffer severely. The aged bear whooping-cough badly. It is peculiarly fatal among negroes. The danger of early or remote tuberculosis lends especial importance to this disease. Death may occur during a paroxysm from intracranial hemorrhage or asphyxia, but such accidents are exceedingly uncommon. The prognosis is to some extent modified by the frequency as well as by the severity of the paroxysms. Cases run a more favorable course in summer than in winter. Reliable general statistical facts relating to the mortality are not available. Many of the milder cases never come under medical observation. In foundling asylums and children's hospitals the death-rate may exceed twenty-five per cent.

X. MUMPS.

Epidemic Parotitis.

Definition.—An acute infectious febrile disease, prevailing in limited epidemics, and characterized by inflammation and enlargement of the salivary glands, especially the parotid.

Etiology. — Predisposing Influences. — Mumps is a wide-spread disease and is usually endemic in large cities. Sporadic cases occur and become foci of circumscribed outbreaks which run a lingering course of months or, in some instances, of a year or more. The infecting principle is much less readily transmitted than that of many of the contagious diseases and the congenital immunity much more common. In general practice extensive epidemics are infrequent, but when the disease appears

in reformatory institutions and schools a large proportion of the inmates usually contract it. The cases are more numerous in the spring and autumn than at other seasons. Mumps is peculiarly a disease of childhood and adolescence. It is not common in early infancy nor after the twentieth year. More boys than girls suffer in a ratio estimated as high as two to one.

Exciting Cause.—The specific cause has not been demonstrated. The disease is directly transmitted by personal contact. Rare instances have been observed in which the contagion has been indirectly transmitted by a third person or by fomites, especially clothing. Two views may be entertained as to the mode of infection. The first is the one generally accepted, namely, that the pathogenic principle finds its way from the mouth to the glands along the course of the salivary ducts and, as the parotid is usually involved, through the duct of Stenson; second, that the infection is a general one, to which certain anatomical structures, as the salivary glands and, in particular, the parotid gland, especially react. The occasional occurrence of inflammation of the testes, and of the ovaries and mammæ in the female, and the definite incubation and typical course of the disease support the latter view.

Fig. 234.—Mumps.—Cotton.

Symptoms.—The period of incubation varies from fourteen to twenty-one days. In rare instances it has appeared to be shorter. Prodromes are commonly absent. In mild cases the swelling and associated local symptoms constitute the earliest manifestations. In severer cases more or less pronounced constitutional disturbance, with shivering, vomiting, and moderate fever, 100°–101° F. (37.8°–38.3° C.), characterize the invasion, which is abrupt and precedes the local inflammation by about twenty-four hours. In severe cases the temperature may reach 103°–104° F. (39.5°–40° C.). A feeling of tension with soreness is felt just below one ear, more commonly the left. Upon examination slight swelling may be observed, which increases until, in the course of forty-eight hours, it reaches its maximum. The parotid is now greatly enlarged and the adjacent tissues of the neck and often of the side of the face tensely œdematous. The skin is glossy, hard to the touch, its folds are obliterated, and, commonly, by reason of interference with the circulation by pressure, white in color. It pits only slightly upon pressure. The swelling occupies the lateral region of the neck between the jaw and the mastoid process, extending upward to the zygoma and downward and forward toward the clavicle and the median line. Its extent varies with the intensity of the attack. The ear is pushed upward, and its lobule, which occupies the centre of the swelling, is sharply pushed outward. In almost all instances the other side is affected in a day or two, sometimes not for several days or

until the inflammation upon the side first affected has subsided. Very often the swelling of the second gland is so slight that it can only be detected upon close scrutiny. The disfigurement is marked and when both sides are affected the patient may be scarcely recognizable. In some instances the other salivary glands are involved, and several cases have come under my observation in house epidemics in which the submaxillary glands have been inflamed while the parotids have remained unaffected. The sublingual glands and the lachrymal glands may also be involved. Movements of the jaw, the act of deglutition and, in severe cases, even phonation are attended with difficulty and pain. There is trouble in taking any form of nourishment, even liquids. The fetor is often extreme. Movements of the head are restricted and in order to relieve tension there is flexion of the neck toward the affected side. The salivary secretion is usually diminished, exceptionally increased. Its reaction may be acid. Slight deafness and earache often occur, and in rare instances otitis media. Permanent deafness, usually one-sided and complete, without otitis media, has been observed. This condition develops suddenly with nausea, vomiting, vertigo and a staggering gait. It is probably due to a lesion of the labyrinth. Leukocytosis is exceptional, except in severe cases. After from five to ten days the swelling gradually subsides, the stiffness and impairment of movement disappear, and normal conditions are restored. Local desquamation may occur. The glandular inflammation undergoes resolution without abscess formation. If suppuration occurs it is due to a mixed infection and must be regarded as a complication. The pus may be evacuated externally or may burrow in the tissues of the neck. Secondary pyothorax or pyopericardium may follow with or without general sepsis.

Orchitis occurs in about one-third of the cases after puberty. In infancy and childhood it is exceedingly rare. It usually affects one testicle only, occasionally both. Weight, swelling, and pain are the symptoms. The testicle may be enormously enlarged. Epididymitis is not common. There may be effusion into the tunica vaginalis, scrotal œdema, and a mild mucopurulent urethral discharge. Atrophy may result and, when both testicles are involved, loss of the procreative function. Even with some atrophy of both testicles functional power may be retained. In adolescents and young adults great anxiety upon this question arises during the attack. Mastitis may occur in boys. In females also, usually after the age of puberty, enlargement and tenderness of the breasts, pain and tenderness in the ovaries, hæmatoma of the labia, or a vulvovaginal discharge may occur. These phenomena are very uncommon. Thyroid enlargement is extremely rare.

Exceptionally the symptoms are very severe. High fever may be accompanied by vomiting, delirium, and sleeplessness. Great exhaustion may result. As a rule the patient is not seriously ill. The constitutional disturbance comes to an end within a week; the local symptoms more slowly.

Relapse is rare. The attack confers an immunity which is practically permanent.

Complications and Sequels.—The frequent involvement of the generative organs and the fact that it sometimes precedes the parotitis or occurs without it compel the recognition of these local inflammations as

incidental manifestations of the disease rather than complications. The frequency of actual complications varies in different epidemics. The fatal cases are frequently associated with meningeal symptoms. Hemiplegia, coma, and acute mania may occur. Among the rare complications are albuminuria, nephritis with uræmic accidents, polyarthritis, endocarditis, facial palsy from pressure, peripheral neuritis, and hemiplegia. The common sequels have been already indicated. The inflamed glands may not undergo resolution but remain enlarged and hard. Ptyalism or xerostomia may persist for some time. Parotid bubo is very rare in idiopathic mumps. Local gangrene may occur. Deafness may be permanent. Optic atrophy is among the rarest of sequels.

Diagnosis.—The DIRECT DIAGNOSIS of mumps is under ordinary circumstances easy. The location of the swelling in front of the ear and below it, and the abrupt displacement of the lobule outwards, together with the circumscribed outline at first corresponding to that of the parotid, are important anatomical considerations. Mumps is a primary affection and very rarely goes on to suppuration. The relative rapidity with which the swelling develops and subsides is characteristic of mumps.

DIFFERENTIAL DIAGNOSIS.—1. Parotid bubo. The glandular inflammation is secondary to some acute infection or sepsis and commonly proceeds to multiple or general abscess formation. This condition is very rare in childhood. 2. Acute cervical adenitis. The swelling is below the angle of the jaw. It does not at any time correspond to the outline of the parotid; nor has it the location of the submaxillary glands. It may be tuberculous or secondary to tonsillar or peritonsillar infection. Irregular contour, redness, the absence of involvement of the generative glands in all cases, and the absence of a definite, self-limited course are to be considered. 3. Hodgkin's disease is a chronic affection of the lymphatic glands. The salivary glands are not involved. 4. Abscess from disease of the jaw in dental caries gives rise to swelling localized in wholly different positions from that of the parotid and is not characterized by definite constitutional phenomena or transmissibility. 5. Gonorrhœal orchitis has a definite history and upon examination of the discharge the gonococcus is found. Doubts can only arise when the inflammation of the testes precedes or occurs coincidently with the parotitis.

Prognosis.—Mumps, in the vast majority of cases, is a mild disease and terminates in complete recovery in a short time.

XI. INFLUENZA.

Epidemic Catarrhal Fever; La Grippe.

Definition.—An acute, infectious, pandemic disease, caused by the bacillus of Pfeiffer. It is characterized by catarrh of the mucous membrane of the respiratory tract, less frequently of the digestive tract, by quickly on-coming debility and nervous symptoms. There is a tendency to complications, especially pneumonia. The general outbreaks are followed for some years by the local epidemic or endemic occurrence of the disease.

Etiology.—Predisposing Influences.—When the disease invades a community, a large proportion of the population is attacked without distinction. Previous illness affords no protection. Aged and infirm persons and those of nervous temperament are peculiarly liable to suffer, but the robust possess no immunity. All races and dwellers in every clime are liable to the disease. Adults are attacked earlier than children, and in some epidemics the latter have manifested a slight relative immunity. A limited number of persons appear to be immune and there are those who, having passed through a series of outbreaks in safety, finally acquire the disease. An attack of influenza confers no exemption from the disease in subsequent outbreaks, and, independently of relapses, which are frequent, individuals have been known to experience a second attack during the prevalence of the same epidemic. The disease bears no relation to known atmospheric conditions. It may prevail at any season of the year. It follows lines of travel and advances at about the ordinary rate of commercial intercourse. The duration of the outbreak in a community is from four to eight weeks, exceptionally a longer time. The epidemics rapidly reach their height and usually subside almost as suddenly as they begin. In large cities influenza makes its appearance at the same time in several different localities and spreads from these as foci of infection throughout the community. Influenza bacilli have been found in the sputum from tuberculous vomicæ and bronchiectatic cavities, and patients suffering from these conditions may be influenza carriers.

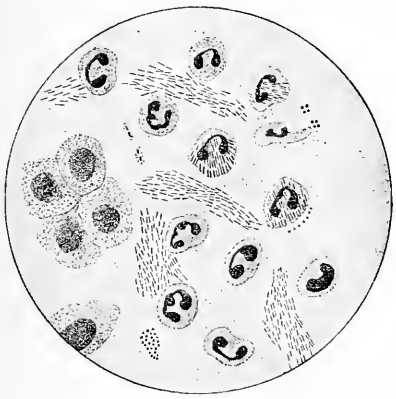


Fig. 235.—Bacillus influenzæ in sputum.

Exciting Cause.—Pfeiffer—1892—isolated from the nasal and bronchial secretions of patients suffering from influenza a bacillus now recognized as the cause of the disease. It occurs in great numbers in the nasal secretions and is frequently seen in the sputum in almost pure culture. It persists in the secretions for some time after the symptoms have subsided.

The disease is readily transmissible by direct contact and fomites; also to short distances by the atmosphere. Influenza bacilli are destroyed by drying, and rapidly perish in water. They enter the body with the inspired air. Pfeiffer, from a study of the biological characters of the influenza bacillus, concludes that its development outside the human body—that is to say, in the ground or in water—is impossible; that its dissemination when dry can take place only to a limited extent, and that the contagium is, as a rule, transferred by the recent moist secretion from the nasal and bronchial mucous surfaces of influenza patients—droplet infection.

Symptoms. — Influenza presents the greatest variations as regards intensity, from a trifling indisposition to an illness of the gravest kind. In every epidemic the majority of the sufferers manifest the disease in a mild form, very many in a rudimentary form. The symptom-complex

is extremely variable and greatly modified by complications and sequels. The period of incubation varies from a few hours to three days. Prodromal symptoms are rare. The onset is abrupt, marked by chilliness or a chill which may be repeated. There is fever, headache (usually intense), with pain back of the eyeballs, severe pain in the back, limbs, and joints, and a general feeling of muscular soreness with tenderness upon pressure. These symptoms are accompanied by mental and physical depression, with malaise and restlessness. The circulation is depressed, the spleen slightly enlarged. In a considerable proportion of the cases catarrhal phenomena do not occur and the attack consists of a fever-storm with its associated phenomena, together with rapidly developing asthenia, more or less profound. The cases may in general be grouped as mild and severe.

In mild cases the chill may be slight or absent altogether. Headache and muscle pains are moderate. There is a sense of weariness upon effort,

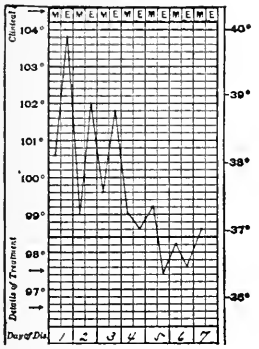


FIG. 236.—Influenza—remittent type.

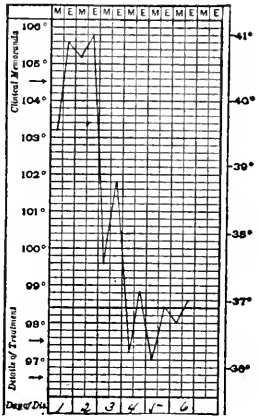


FIG. 237.—Influenza—interrupted crisis.

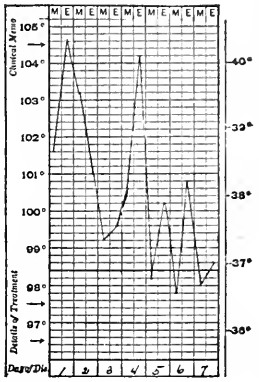


FIG. 238.—Influenza—intermittent type.

disinclination for affairs, some difficulty in fixing the attention. Coryza, erythematous angina, and a tickling cough occur. The fever is usually slight, the temperature not rising above 102° F. (39° C.). Many of the patients suffering from influenza in this form are able to continue their ordinary avocations. No great intensification of the symptoms is necessary, however, to compel them to betake themselves to bed.

In the severer cases the chill is more marked or the shivering more prolonged. Fever is rapidly established, the acme being reached within twenty-four or thirty-six hours. The temperature may rise to 104° or 105° F. (40°–40.5° C.). Sensations of heat alternate with chilliness. There is pain in the orbits and at the root of the nose. Coryza is severe. Epistaxis is occasionally observed. The throat is sore; there are tickling sensations in the upper air-passages, hoarseness, and sometimes dyspnœa. The cough is paroxysmal, distressing, and at first unproductive. Chest pains and stitches in the side also occur. The pulse may be full and compressible; more commonly it is feeble, small, and irregular. It is as a rule only moderately increased in frequency. In some cases there is slight

blueness of the lips and finger-tips. The patient is distressed by want of sleep. At the end of four or five days the febrile symptoms decline rapidly, less commonly gradually. The defervescence is often accompanied by copious sweat, spontaneous diarrhœa, increased flow of sedimentary urine, and considerable amelioration of the subjective symptoms. The catarrhal symptoms outlast the fever two or three days, but cough and expectoration may persist for some time. Cutaneous hyperæsthesia is often present and areas of burning pain in the skin occur. Neuralgias may develop during the attack and persist for a long time.

Symptoms referable to the nervous system may dominate the clinical picture. In other cases gastro-intestinal symptoms are conspicuous. The attack may develop abruptly with symptoms like those of cholera morbus. Finally, cases occur in which there is no marked localization of the infectious process. The patient suffers from fever and great depression and simultaneous implication of the respiratory, circulatory, nervous, and gastro-intestinal systems.

Herpes is common. Urticaria and purpura have been observed. The sense of smell is often lost and that of taste impaired or perverted. The hearing is blunted. Febrile albuminuria is not uncommon in the severe cases. A mild anæmia develops in grippe. Leucocytosis does not occur in uncomplicated cases.

Attempts have been made to arrange the cases of influenza in different groups, and in theory a thoracic, cardiac, gastro-intestinal, and nervous variety may be recognized. In practice, however, various described types merge into each other, and are so modified by individual peculiarities of the patient and by complications which arise in the course of the attack that there is but little advantage in referring particular cases to theoretical categories.

The duration of the milder forms of influenza is from one to three or four days. In well-developed cases without complications convalescence sets in between the fourth and seventh days. Severe cases with complications may be protracted for several weeks. Relapses occur in about 10 per cent. of the cases. If the fever continues beyond the seventh or eighth day it will usually be found upon careful examination to be due to some complication. The temperature curve of influenza not infrequently merges into that of a complicating bronchitis, bronchopneumonia, or croupous pneumonia.

Complications and Sequels.—Among the complications, intense bronchitis, implicating the large and small tubes and giving rise to a prolonged symptomatic fever, may occur. This bronchitis has no special peculiarities. The sputum may be abundant and thin, or may be of a greenish-yellow color and nummular. It is sometimes bloody. Bronchopneumonia is not uncommon, especially in children and aged persons. It may be due to the influenza bacillus or to mixed infection. It constitutes a serious complication and is a frequent cause of death. Influenza pneumonia may occur at any time during the course of the attack. Its symptoms are frequently obscure and its course irregular. Extensive involvement of the lung may take place without great rise of temperature. Croupous pneumonia is less common. Abscess or gangrene of the lung may follow the pneumonia of grippe. Pleural effusion is not an uncommon complication

and empyema may occur. Pulmonary tuberculosis may develop after an attack of influenza, or, if already present, it is usually aggravated. Endocarditis and plastic or purulent pericarditis may occur in connection with pneumonia or independently of that complication.

Among complications relating to the nervous system meningitis, encephalitis, and brain abscess are to be mentioned. Peripheral neuritis not uncommonly develops during the course of the attack. Headache, insomnia, and neuralgia are common sequels. Forms of neurasthenia occur. Hysteria and chorea have been noted, and psychic disorders, as melancholia and the insanities of malnutrition.

Otitis media constitutes one of the more distressing complications and sequels of influenza. Rapid disorganization of the structures of the middle ear may give rise to permanent deafness. Persistent vertigo may follow influenza. Conjunctivitis is frequent and may be severe. Iritis and optic neuritis are rare sequels. I have seen severe and protracted xerostomia develop after defervescence. Cardiac symptoms are common and distressing. They consist of heart consciousness, precordial pain, breathlessness and faintness upon effort, and unsatisfactory sleep. The physical signs are those of an enfeebled and irregular heart. Arrhythmia, tachycardia, and bradycardia are common. These symptoms are to be ascribed in part to the disturbed nutrition of the heart muscle and in part to the derangements of the cardiac innervation. An attack of influenza has appeared in some instances to be the starting-point of pernicious anæmia. Less common complications and sequels are parotitis, nephritis, phlebitis, venous and arterial thrombosis.

Diagnosis.—Direct Diagnosis.—During a pandemic it is unattended with difficulty. The progress of the outbreak, the number of individuals attacked nearly at the same time or in quick succession, the profound asthenia, and the prominence of the nervous symptoms serve to distinguish it from other epidemic diseases. Bacteriological diagnosis can be made by an examination of the bronchial sputum.

Differential Diagnosis.—*Non-specific Influenza.*—The differential diagnosis between influenza and non-specific catarrhal affections rests upon the pandemic or epidemic prevalence of the former, great prostration, and prominence of the nervous symptoms. The relation of these two diseases is analogous to that between *cholera Asiatica* and *nostras*. The diseases designated by the term *influenza* may be divided into: (1) pandemic *influenza vera*, caused by the bacillus of Pfeiffer; (2) endemic-epidemic *influenza vera*, recurring from time to time locally after the pandemics, caused by the same infecting agent; (3) endemic *influenza nostras*—pseudo-influenza, catarrhal fever—sometimes miscalled grippe—a disease *sui generis*. The infecting micro-organism is not known. *Enteric Fever.*—In the gastro-intestinal form the malaise, headache, dulness of hearing, mental depression, fever, epistaxis, a coated tongue, tender belly, and diarrhœa may suggest enteric fever. An attack of influenza in uncomplicated cases runs its course before the time at which splenic tumor and rose spots establish the diagnosis of enteric fever. The occurrence of influenza during the period of incubation of enteric fever may add to the difficulties of diagnosis. Bacteriological methods and especially the Widal test are necessary

in doubtful cases. *Cerebrospinal fever* has prevailed during some epidemics of influenza. The occasional occurrence of cases of influenza marked by painful retraction of the muscles of the back of the neck and vomiting renders the differential diagnosis between these two affections difficult. Nor is the fact to be overlooked that meningitis occurs as a complication of influenza. *Dengue* closely resembles influenza. Each of these diseases occurs in abruptly developing pandemics affecting almost all the inhabitants of the regions invaded. They resemble each other in the frequency of relapse, liability to repeated attacks during the same outbreak, the fact that they are not self-protective, in the want of accord between the gravity of the symptoms and the low death-rate of uncomplicated cases, the suddenness of the attack, intensity of the pains, and the high degree of mental and physical depression. Influenza lacks, however, the cutaneous manifestations, the remission in the course of the fever, and the tendency to arthritis seen in dengue. It differs also in the liability to serious complications and in prevailing in all climates.

Prognosis.—Death is rare in uncomplicated cases except at the extremes of life. The very young bear influenza badly, the aged bear it worse. Previous disease is unfavorable. Individuals suffering from chronic bronchitis, emphysema, myocarditis, and nephritis offer diminished resistance. Exhausting diseases increase the danger of the attack. Cases attended by very severe symptoms usually recover unless the patient be very young or very old or the subject of some complicating malady. The prognosis is greatly modified by the character of the prevailing epidemic. In some epidemics the death-rate has been low and the mortality from other diseases only slightly increased. More commonly the death-rate of endemic affections is much increased and in some epidemics influenza has been attended by a high direct death-rate. In the epidemic of 1918 more than 5000 deaths occurred in Philadelphia in a single week.

XII. DENGUE.

Definition.—A pandemic infectious disease of tropical and subtropical climates, characterized by a febrile paroxysm with recurrence, intense pains in the joints and muscles, and an early erythematous and a late polymorphous eruption.

The popular term *break-bone fever* denotes the atrocious character of the pain.

Etiology.—Predisposing Influences.—Dengue first excited general attention by its epidemic prevalence in the West India Islands in 1827. Benjamin Rush observed an outbreak in Philadelphia in 1780. Dengue is, in the strictest sense, a pandemic disease. No other disease, with the exception of influenza, prevails so widely and attacks so large a proportion of the population. Equally remarkable is its rapidity of diffusion. In Galveston in the epidemic of 1897, 20,000 persons were attacked in the course of two months. Dengue is a disease of warm climates and of warm seasons. When it has occurred in the summer in temperate climates it has disappeared upon the appearance of frost. The recent experimental investigations of Ashburn and Craig led them to believe that dengue is not a contagious disease, but that it is infectious in the same manner as yellow

fever and malaria and that the mosquito at fault is probably *Culex fatigans* (Wied). The liability is universal. Neither age, sex, nor occupation confers immunity. The outbreaks chiefly affect cities, less generally the open country. To this statement, however, there have been many exceptions.

Exciting Cause.—No organism, either bacterium or protozoon, can be demonstrated in either fresh or stained specimens of dengue blood with the microscope (Ashburn and Craig). The pathogenic principle has been shown to be a filterable virus.

Symptoms.—The period of incubation varies from three to five days. At the beginning and at the height of epidemics it has not, in some cases,

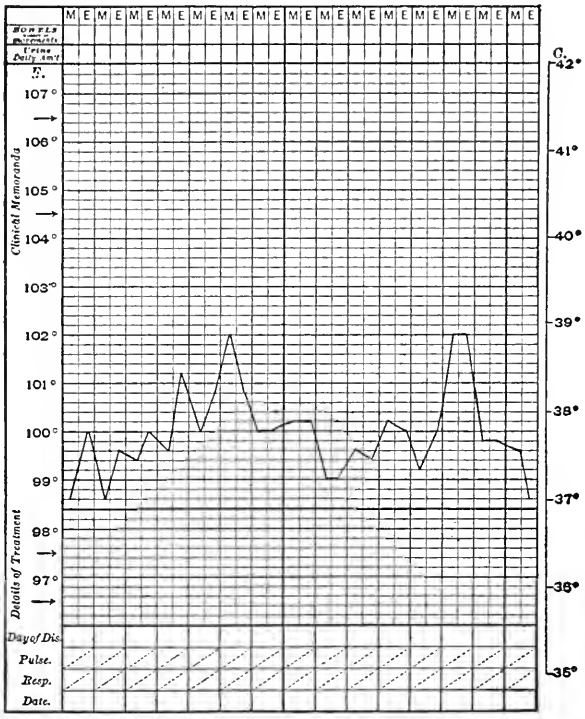


Fig. 239.—Dengue.—Ashburn and Craig.

exceeded a few hours. The invasion is abrupt. Prodromes are unusual; when present they consist of lassitude, headache, a furred tongue, loss of appetite, muscular soreness, and chilliness. The attack sets in with intense headache, backache, and severe pains in the joints and muscles. The affected joints are swollen, and the face and neck flushed and turgid. Movements are executed with pain and difficulty. Conjunctivitis, swelling of the eyelids, intolerance of light, and stiffness of the muscles of the eyeballs are common. The tongue is heavily coated, and there is epigastric distress which is followed by nausea and vomiting. Appetite is lost and the bowels are constipated. Thirst is not a prominent symptom. The temperature rises rapidly, often reaching by the end of the first twenty-four hours 106.7° to 107.6° F. (41.5° to 42° C.). The pulse is tense and frequent, 120 to

140. The breathing is rapid, the skin hot and dry. Confusion of thought and delirium occur, and in children the attack may begin with convulsions. An erythematous rash appears in many of the cases.

The duration of the first febrile paroxysm is variable, lasting from a few hours to several days, the average being about three days. Defervescence is commonly sudden and is often attended with critical discharges, such as profuse sweating, epistaxis, or diarrhœa. The eruption now disappears, the skin becomes moist, and there is an amelioration of the pains in the muscles and joints.

The afebrile period lasts two or three days. In some cases it does not occur, or is so brief as to be overlooked. There are cases in which the temperature falls but does not quite reach the normal. Notwithstanding the great relief which the patient experiences, there remain some headache and stiffness of the muscles and joints. At the expiration of several hours, more commonly of two or three days, a second febrile paroxysm sets in. The symptoms are much the same as those of the initial paroxysm but less intense. An eruption of variable character develops at this stage of the disease. It is sometimes macular like the rash of measles, or diffuse and erythematous like that of scarlet fever, or papular. Sometimes it resembles an urticaria, and there are cases in which vesicles occur. Appearing in many instances first upon the hands and feet, this eruption generally invades the greater part of the surface of the body. In other cases it is limited to certain areas. It is attended by distressing itching, and gradually fades after two or three days, being followed by a branny desquamation. The duration of the second paroxysm is from two to three days. Defervescence is gradual; the acute symptoms disappear and the patient enters upon convalescence much enfeebled, the muscular pains and stiffness of the joints often lasting for a considerable time. The small and large joints are alike involved, many being affected at the same time or in rapid succession. The muscles are also stiff and sore, and this, with the swollen condition of the integuments, greatly increases the difficulty and awkwardness of movement, especially in the fingers and hands. In severe cases the mucous membrane of the mouth, throat, and nose is inflamed. The secretion of saliva is sometimes increased and the salivary glands, in particular the parotid, are swollen and tender. The superficial lymph nodes about the angle of the jaw and in the groins are in some cases enlarged.

The Blood.—The white corpuscles are diminished in number and there is a relative increase in the small mononuclear variety. The characteristic blood findings are: (1) the absence of a demonstrable protozoon; (2) leucopenia; (3) diminution of polymorphonuclears, and (4) a striking variation in the percentage of other leucocytes at different periods of the attack (Stitt).

The urine during the access of fever is scanty and of high color. With the crisis the quantity is increased. Albuminuria is not usually present.

Diagnosis.—The DIRECT DIAGNOSIS is not difficult. No other disease spreads with such rapidity through a community and attacks so large a proportion of the inhabitants.

DIFFERENTIAL DIAGNOSIS.—Influenza, see p. 702. From acute articular rheumatism dengue differs in its course and duration as well as

in the eruptions which attend it; from scarlet fever and measles, in everything except the occurrence of the rashes, which resemble the exanthems of these diseases only in the most superficial way; from relapsing fever in all things except its course, and from yellow fever in many important particulars, among which its extremely low death-rate, the absence of jaundice and black vomit, the infrequency of hemorrhage and albuminuria, and the correlation between the pulse-rate and the temperature are of diagnostic importance. Yet upon these very points experts have failed to agree in the differential diagnosis, as in the State of Texas during the epidemic of 1897. The difficulty is increased by the fact that yellow fever and dengue have the same habitat and very often prevail side by side.

Prognosis.—Dengue is seldom fatal. A few fatal cases have been noted in extensive outbreaks; but in many epidemics in large cities not a single death has occurred.

XIII. DIPHTHERIA.

Definition.—A specific infectious disease, caused by the Klebs-Löffler bacillus and characterized by a fibrinous exudate, usually situated upon and in the mucous membrane of the upper respiratory passages, and by serious constitutional symptoms due to toxins formed in the local lesions.

Clinically the term diphtheria is applied to cases of pseudomembranous inflammation in which the presence of the Klebs-Löffler bacillus can be demonstrated. Cases of pseudomembranous inflammation, which present similar local and constitutional features, but in which Klebs-Löffler bacilli are not present, are designated *pseudodiphtheria* or *diphtheroid angina*.

Etiology.—Predisposing Influences.—Diphtheria is widely distributed but is especially a disease of temperate climates. The general prevalence is more extensive in winter and it is probable that the frequent occurrence of catarrhal inflammation of the pharyngeal mucous membrane constitutes a definite predisposing factor. Severe outbreaks are occasionally encountered in summer. Neither altitude nor the constitution of the soil exerts a special influence. Diphtheria is endemic in cities and towns, where it frequently becomes epidemic, but this is also the case in rural districts, where the population is scattered, and among hamlets and farms it often rages with extreme virulence. The affection spares no class in a community. Rich and poor alike contract it. Crowding, uncleanliness, and neglect of sanitary laws favor the spread of the disease among the poor; therefore epidemics are much more common among the poor than among well-to-do and well-advised persons. These elements of predisposition are fully explained by the fact that diphtheria is a readily transmissible disease. Infants in the first year of life are rarely affected. The period of greatest liability lies between the second and the fifteenth years. The mortality is especially great between the second and the fifth years. The disease may occur at any age and it not infrequently happens that the nurse or a parent contracts the disease from a child, and many physicians have lost their lives from diphtheria contracted in the discharge of duty. In later childhood and adolescence girls are attacked more frequently

than boys. The diminished resistance to pathogenic influences on the part of the local mucous membrane which accompanies chronic nasopharyngeal catarrh, adenoid vegetations, hypertrophied tonsils, and laryngitis constitutes an especial predisposition to the disease. Certain acute febrile infections, such as measles and scarlet fever, act in the same way. The pseudomembranous inflammation of the acute diseases is more frequently diphtheroid and due to streptococcus infection.

The attack does not confer immunity; on the contrary, certain persons manifest a peculiar susceptibility. There are on the other hand persons who appear to be possessed of a natural immunity and do not contract the disease upon exposure nor, in some instances, even when the bacilli are present in their throats.

Schick's Test.—See Vol. I, p. 624.

Exciting Cause.—The Klebs-Löffler bacilli are non-motile; they vary in length from 2.5 to 3 μ and from 0.5 to 0.8 μ in breadth, and present different forms, some of which are pointed, others blunt and rounded at the ends, and less frequently forms that are irregular and branching. They are very tenacious of life and have been grown in culture after a non-parasitic existence under varying conditions for periods measured by weeks and months. Attached to clothing, bedding, articles of furniture, even the walls and floor of the room occupied by the patient, they maintain their pathogenic power. Milk serves as a culture media in which they grow rapidly without altering its appearance. The bacilli are found in large numbers in the false membrane of cases of true diphtheria. In this situation they grow and multiply, being rarely found in the blood during life. In the lesions of the bronchopneumonia of laryngeal diphtheria they may be the predominant organism. After death they are occasionally present in the blood and viscera. They vary greatly in virulence. Guinea-pigs, the most susceptible of laboratory animals to the poison, are used for determining the intensity of cultures. Non-virulent bacilli are sometimes spoken of as the pseudobacillus of diphtheria—a misnomer. The pseudodiphtheria bacillus or *Bacillus xerosis* resembles the Klebs-Löffler bacillus morphologically, but differs from it in certain cultural peculiarities and being non-pathogenic. The biological relationship of this organism to the Klebs-Löffler bacillus has not been determined.

Fig. 240.—Bacillus diphtheriæ from culture on Löffler's blood-serum.

The Klebs-Löffler bacilli may be present upon the mucous membrane of cases which show no membranous exudate and present the clinical picture of a lacunar tonsillitis or a simple erythematous angina. They are very often found in the throat and nose of individuals who have passed through an attack of diphtheria and are fully convalescent. Under these circumstances they may persist for weeks or months, during which time

the patient may communicate the disease to others or pass through one or more fresh attacks himself. They are frequently isolated from the throats of healthy persons, both the nurses and attendants upon the sick and others living in the same house with the patient or convalescent—bacillus carriers. The organisms have been found in the hair and clothing of nurses and in the dust of diphtheria wards. The disease may be communicated by means of infected milk.

The foregoing facts shed considerable light upon the endemic and epidemic prevalence of the disease, as well as the occurrence of sporadic cases. They enable us to understand the persistence of diphtheria in localities, the occurrence of house and neighborhood epidemics, the simultaneous occurrence of scattered cases in a school district, and the development of cases in patients long confined in hospital wards, after visiting days.

The Toxin.—The general symptoms are caused not by the bacilli but by toxins formed by them. Susceptible animals may be rendered immune by the injection of progressively stronger doses of attenuated cultures of the bacilli or increasing quantities of the toxin. By suitable treatment in this manner large animals, as the horse, may be rendered to a high degree immune.

Symptoms.—The period of incubation varies from two or three to seven days. The onset is marked by slight chilliness and sometimes in young children by convulsions. The signs of a febrile infection, elevation of temperature,—102°–103° F. (38.9°–39.5° C.),—malaise, backache, and muscle pains follow. These symptoms vary greatly in intensity. Older children and adults complain of sore throat. An examination of the fauces must be made as a matter of routine in infants who are taken acutely ill. The pseudomembranous exudate rapidly forms upon the mucous surfaces of the tonsils and adjacent parts and shows a marked tendency to spread upwards to the nasopharynx, the nasal chambers and accessory sinuses, the tear duct and the Eustachian tube, or downwards to the epiglottis, larynx, trachea, bronchi. Much less frequently the œsophagus, stomach, and duodenum are invaded. Accidental infection of the vulva and vagina, the ear, conjunctivæ, and wounds occurs. The larynx is primarily involved in a large proportion of the cases.

According to the local lesions the following groups of cases are to be considered:

1. Faucial Diphtheria.—The mucosa is at first reddened and there is difficulty in swallowing. The membrane usually first appears upon one tonsil and, in the course of a few hours, without bridging across, upon the other. After some hours, or it may be a day or two, it has in neglected cases covered the tonsils and spread to the half-arches, the soft palate and uvula, and to the pharynx—pharyngeal diphtheria. Meanwhile, the tonsils are enlarged and the soft palate and uvula are swollen, reddened, and œdematous. The membrane, at first whitish, soon assumes a gray or dirty yellowish-white color. As a rule the patch or patches are distinctly marginate and surrounded by a border of red deeper than that of the general mucosa. The membrane, when forcibly detached, leaves an eroded surface with punctate bleeding, upon which a fresh pellicle soon appears. The

lymph-nodes about the angle of the jaw are swollen and tender. Variations in the character of the exudate occur. It may be throughout punctiform and restricted to the tonsils, or punctiform at the outset but rapidly becomes membraniform, and extends. Again the exudate may be pultaceous rather than pseudomembranous. Finally, there are cases with acute erythematous angina and constitutional symptoms in which no membrane is present but virulent Klebs-Löffler bacilli are found in the secretions. The breath of the patient has a fetid, sickening odor, which surrounds his person and permeates the atmosphere of the room.

2. Nasal Diphtheria. — The Klebs-Löffler bacilli are frequently found in the nasal secretions when the exudate has invaded the pharynx, although no membrane is present in the nasal chambers. When membranous exudate is present two conditions occur. In the first the nares are occupied by a thick, tough membrane, which rarely extends to the adjacent parts and in which Klebs-Löffler bacilli are present, but the constitutional symptoms are very slight or altogether absent. The disease shows very little tendency to affect other children in the family.

The second and far more common form of nasal diphtheria may be primary, but usually arises in the course of the attack by extension from the pharynx or autoinoculation by way of the nostrils. Exceptionally the symptoms are mild, but in the majority of cases both the local and constitutional symptoms are most intense—a fact attributed to the abundant supply of lymph-vessels to the mucous membrane of the nose and consequent free absorption of diphtheria toxin.

3. Laryngeal Diphtheria.—The term membranous croup was at one time used without distinction to designate all forms of membranous laryngitis. This misleading and dangerous custom has fortunately passed away. More than four-fifths of such cases in large series of statistics have shown the presence of Klebs-Löffler bacilli. In a considerable proportion of the rest the result, for various reasons, has been doubtful, while in the small remainder other organisms, chiefly streptococci, have been found. While the clinical symptoms are practically the same, the differential diagnosis can readily be made by laboratory methods, and the membranous laryngitis associated with the Klebs-Löffler bacillus is called laryngeal diphtheria, while that in which other organisms are exclusively present is known as diphtheroid laryngitis or pseudodiphtheritic laryngitis. The latter affection is rarely a primary disease, but usually arises as a complication in the course of some acute disease—scarlet fever, variola.

The local symptoms of laryngeal diphtheria are at first those of an acute laryngitis, with hoarseness and a rough, so-called "croupy" or laryngeal cough. In the course of twenty-four or thirty-six hours the patient—usually a child—suddenly becomes worse, with symptoms of laryngeal stenosis—dyspnœa, slight cyanosis, rapid pulse, aphonia, brassy cough, and restlessness. These symptoms, which commonly develop at night, are at first paroxysmal, with intervals of quiet breathing and sleep. In favorable cases, after two or three paroxysms without marked dyspnœa or cyanosis, the child falls asleep and awakes in the morning greatly improved. Not rarely the attack recurs upon the succeeding night with more intensity.

The respiratory obstruction, which is at first due in part to laryngeal spasm, with the increasing exudate soon ceases to be paroxysmal and becomes continuous with exacerbations and remissions. Inspiration and to a greater extent expiration are increasingly difficult. The auxiliary respiratory muscles are brought into play, the lower intercostal spaces and epigastrium show inspiratory retraction. The voice is reduced to a husky whisper. The cyanosis of the lips and finger-tips becomes more intense. There is urgent air hunger and after a period of extreme restlessness the patient sinks into a semiconscious listlessness, with general relaxation and a freely perspiring skin, only to start up again in the course of a few minutes, tossing about and struggling for air. Occasionally in a severe paroxysm of cough shreds of membrane are coughed up with great temporary or even permanent relief. In other cases, a fold of detached membrane becomes lodged in the glottis and is followed by fatal asphyxia. The fatal issue is, as a rule, preceded by increasing dyspnœa and cyanosis, a period of distressing jactitation, coma, and slight, shuddering convulsions. Pharyngeal exudate may be present, the membrane invading the larynx from above—descending croup; the invasion being from the larynx upward—ascending croup. In many of the cases the membraniform exudate is situated wholly within the larynx, where it may be seen upon laryngoscopic examination—a procedure usually attended, however, with great practical difficulties. If the duration of the attack be prolonged, bronchopneumonia occurs. This complication may be due to an extension of the bronchitis, caused by retained secretions, to the finer bronchial tubes—secondary infection—or to an infralaryngeal extension of the exudate along the trachea and into the bronchial tubes—a true diphtheritic tracheobronchitis. Thus arises respiratory obstruction at two anatomical levels, namely, at the larynx and in the smaller bronchial tubes, a condition often difficult of recognition because of the diminution of tidal air and consequent fainter vesicular sounds and small mucous râles on the one hand, and the loud laryngeal stridor and coarse tracheal râles on the other; a fact of great practical importance because a successful intubation or tracheotomy, which wholly relieves the obstruction at the upper level, can have no effect whatever upon that at the lower level.

4. Other Sites of the Diphtheritic Exudate.—The conjunctiva may be the seat of a primary or secondary diphtheria. In the latter case the extension is by way of the tear duct or by autoinfection. The symptoms may be those of a catarrhal conjunctivitis, the baciili being present in the secretions, or they may be very serious. The invasion of the middle ear by way of the Eustachian tube may be the occasion of an otitis causing destruction of the tympanic membrane and erosions of the external meatus covered with a characteristic membrane. Vulvar and vaginal diphtheria is occasionally encountered. Diphtheria of the skin occurs in the ordinary forms of faucial and nasal diphtheria when, as is not rarely the case, fissures and abrasions form about the nostrils and corners of the mouth and become infected. The membrane in diphtheria of the anus or genitalia may likewise invade the adjacent cutaneous surfaces. Wounds and ulcerated surfaces in persons suffering with diphtheria are liable to be the seat of an adherent pseudomembrane associated with the Klebs-Löffler bacillus.

The organism may be present in inflamed or necrotic lesions with membrane and, in rare instances, wound infection may occur in the absence of throat affection or traceable exposure to diphtheria cases or fomites. A large proportion of the cases of pseudomembranous inflammation of wounds are due to streptococcus infection or to mixed infection. Local diphtheritic lesions, when severe, are frequently associated with more or less necrosis and gangrene.

In favorable cases the process of separation of the membrane and healing may be observed in faucial diphtheria. After some days the extension of the process is arrested and in the slighter cases the membrane becomes thinner, less distinct at the margins, and gradually disappears. In the more severe forms it appears thicker at the margins, which curl outward from the underlying surface, and separates *en bloc* or by a gradual disintegration. In either case the outlying mucous membrane loses its redness and œdema and shows rapid and marked improvement. Local ulcerations often persist, which, in healing, may give rise to adhesions of the uvula to a tonsil or of the soft palate in part to the wall of the pharynx, and the like.

Infraglottic membranes separate from the underlying surface in more or less extensive membraniform shreds. Pathologically the conditions differ in the mucous membranes above the glottis, which are provided with a squamous epithelium, and in those below it which have a columnar and ciliated epithelium. In the former the membrane is found not only upon, but also in, the substance of the mucosa, while in the latter it is superficial, involving largely the epithelial surfaces and not causing necrosis of the underlying tissues.

Diphtheritic Toxæmia; the Systemic Infection.—There is, in the majority of cases, a general correspondence between the intensity of the local lesions and the severity of the constitutional symptoms. To this rule, however, there are important exceptions. There may be extensive and intense faucial or nasal membranous inflammation with relatively mild general symptoms, or profound toxæmia with limited and apparently superficial local lesions. It has been assumed in explanation of this discrepancy that certain individuals may be more susceptible to the diphtheria bacillus and others more susceptible to its toxins. It is more in accordance with the known facts to explain these differences by assuming that in some instances the bacilli form a larger amount of more virulent toxin than in others and that severe local lesions are in part due to the action of associated organisms—mixed local infections. This explanation finds support in the fact that the graver symptoms are, as a rule, not at first present but arise later when the local disease is at its height. The severest form is septic diphtheria, the outcome of the simultaneous action of the diphtheria bacilli, streptococci, and saprophytic bacteria which are present in the necrotic lesions.

The general symptoms of the attack of diphtheria are those of a mild or intensely severe general infection. The onset is marked by chilliness, a chill, followed by vomiting, fever of atypical course, headache, and anorexia. The temperature varies not only in different cases but also in the course of the attack in the same case. Often but slightly above normal,

it sometimes reaches 104° F. (40° C.). In the severest cases the temperature is sometimes subnormal. The pulse in severe cases is small, weak, and irregular, and in some of the gravest cases there is bradycardia. In the septic cases with gangrenous lesions the constitutional depression may be extreme, with frequent thready pulse, high fever, and nervous symptoms, or there may be ashen pallor, great enlargement of the superficial lymph-glands, and a subnormal temperature. A leucocytosis is present alike in the mild and moderately severe cases.

The following visceral changes occur: The toxin of diphtheria acts especially upon the heart muscle and the nervous system. The myocardium shows fatty degeneration. Endocarditis is rare and the bacilli have been found in the lesions. Pericarditis is extremely rare. Pulmonary complications are very common and are often the cause of death, especially in laryngeal diphtheria. The most common condition is bronchopneumonia. Klebs-Löffler bacilli and streptococci are often present, but the organism in the greater number of cases is the pneumococcus. The liver, spleen, and kidneys show the parenchymatous changes present in the severe infections.

Complications and Sequels.—*The Heart.*—Irregular action is common. A faint, blowing, systolic murmur is heard in a majority of the cases. Rapid action, associated with gallop rhythm and epigastric pain, and bradycardia are grave symptoms. Acute dilatation due to granular and fatty degeneration may be the cause of sudden death in the course of an otherwise favorable convalescence. *Paralysis* occurs in from 15 to 20 per cent. of the cases. It is usually incomplete. In rare cases it comes on as early as the seventh day, but commonly not until the second or third week and sometimes later. It is more frequent in adults than in children. Diphtheritic palsy may follow cases in which the local and constitutional symptoms are mild. The palate is most frequently involved, the symptoms being speech having the nasal quality and the regurgitation of fluids through the nose in swallowing. Upon inspection the soft palate is seen to be relaxed and immobile upon phonation. Sensation is likewise greatly diminished. The constrictors of the pharynx may be affected. The intrinsic and extrinsic muscles of the eye are also frequently involved. Strabismus, ptosis, and loss of accommodation result. The loss of power may affect a single limb or the arms or legs together. As a rule it is the result of peripheral neuritis, and the limbs are flaccid, with impairment or abolition of the tendon reflexes. Multiple neuritis is common. The paralysis may affect the extensors of the feet or there may be complete paraplegia. When the arms are involved the patient is often unable to help himself. In other cases an acute ataxia, resembling tabes but without the lightning pains and pupillary phenomena, has been observed. This condition may be attributed to the action of the toxin upon the posterior columns and posterior nerve-roots and is analogous to the derangements of coördination experimentally produced in animals by the injection of Klebs-Löffler bacilli or the diphtheria toxin.

The occurrence of albuminuria is common and may be noted as early as the first day of the attack. This early change in the urine must be regarded as a "toxic" albuminuria. The albumin in favorable cases dis-

appears in the course of some days. In cases of greater severity it persists and red blood-corpuscles and epithelial and hyaline casts appear. The condition is that of an acute nephritis. Anasarca, contrary to the course of the renal affection in scarlet fever, is very rarely encountered and the acute nephritis shows very little tendency to become chronic.

Hemorrhage from the local lesions occasionally occurs in the severer cases of faucial and nasal diphtheria. Epistaxis may be the first symptom.

A diffuse erythematous rash occasionally develops early in the course of the disease. Urticaria is by no means infrequent, and petechiæ and purpuric hemorrhages appear in the later stages of the grave cases. Jaundice, as in other septic conditions, is often present in the worst cases.

Pseudodiphtheria.—The diphtheroid affection is rarely transmitted to other patients or the attendants. As a rule the local process is of moderate intensity and the constitutional symptoms, if present at all, are mild. There are, however, cases in which the most intense streptococcus infection is associated with non-diphtheritic membranous inflammation of the throat or nose.

Diagnosis.—Direct Diagnosis.—This rests upon the presence of a false membrane having the characters above described; bacteriologically, upon the presence of the Klebs-Löffler bacillus. There are, however, cases of membranous inflammation in which the Klebs-Löffler bacillus is not present—diphtheroid angina—and the bacillus may be frequently demonstrated in cases presenting the clinical phenomena of an ordinary lamnar tonsillitis or simple tonsillar or pharyngeal catarrh, or in the throats of persons in health. This want of accord between the clinical and bacteriological diagnosis is apparent rather than real. The same thing is seen in other affections, as, for example, tuberculosis. It is a question of the seed and the soil. The bacillus varies in virulence and the individual in power of resistance. The Klebs-Löffler bacillus is the criterion. Membranous inflammations associated with it constitute diphtheria; non-membranous inflammations in which it is present are diphtheritic, as tonsillitis, pharyngitis, rhinitis, and the like. The recognition of the diphtheritic character of many of these milder throat affections marked an important advance in practical medicine.

Bacteriological Diagnosis.—For the positive determination of the true character of an acute throat affection a bacteriological examination is often necessary. The material should be taken from the throat as early as possible in the course of the attack and at a time when no antiseptic, and in particular no mercurial preparation, has recently been applied. An immediate diagnosis may sometimes be reached by making a smear preparation. Cultures require about fourteen hours at the body temperature. If the result is negative the examination must be repeated. When the result is positive the examination should be repeated at intervals of ten days or two weeks until the bacilli are no longer found. Pending the result of the examination every acute sore throat in a child must be regarded as suspicious, and measures of isolation and disinfection instituted without delay.

Therapeutic Test.—Antitoxin Treatment.—In every case of pseudo-membranous angina the physician without awaiting the laboratory

report should at once administer diphtheria antitoxin serum in doses regulated not by the age of the patient but by the severity of the process. The dose varies from 6000 to 8000 units and may be repeated if necessary at intervals of twelve to twenty-four hours. In very grave cases a total dosage of 50,000 or 70,000 may be required. When administered early the serum is followed in the course of a few hours by local and general improvement. The swelling of the faucial mucous membrane subsides, the membrane shrivels and gradually disappears, the sickening odor becomes less intense, the temperature falls to normal, and the pulse loses in frequency and gains in force. Even in apparently hopeless cases improvement and eventual recovery frequently occur. Most remarkable results are seen in laryngeal diphtheria, so that intubation and tracheotomy have become far less common than formerly. The diphtheria antitoxin serum is wholly without effect in pseudodiphtheritic membranous angina.

Differential Diagnosis.—*Pseudodiphtheritic Angina.*—The majority of the cases of diphtheroid throat inflammation are caused by the Streptococcus pyogenes. They are almost always secondary to other infections, as scarlet fever, variola, measles, or pertussis. The local and general symptoms are less intense. Exceptionally they are severe. The bacteriological findings are diagnostic.

Prognosis.—Since the introduction of the antitoxin treatment the mortality has progressively fallen and is now about 10 per cent. Before this era it ranged from 30 to 40 per cent. Of unfavorable omen in individual cases are extensive or gangrenous exudate, sanious discharge from the nostrils and intense, penetrating, sickening stench, a feeble, thready pulse, cold, clammy hands and feet, and petechiæ. The common causes of death are laryngeal obstruction, bronchopneumonia, sepsis, sudden asystolism, paralysis, and uræmia.

Serum Disease; The Allergic Reaction; Anaphylaxis.—There are two groups of cases: (a) Those following the primary inoculation within five or ten days and comprising local redness, generalized erythemas and urticaria, fever and arthralgias—all of brief duration, and (b) those caused by reinoculations performed not in the course of a few hours or days but after intervals of several weeks or many months and characterized by immediate and intense œdema and urticaria, accompanied in certain cases by shivering, cyanosis, convulsions, coma and even death. Laboratory studies have shown that these effects are due to the serum itself. Other proteid substances acting upon susceptible individuals, when reinoculated, ingested or inhaled, are capable of causing similar though less intense phenomena. Severe reactions are liable to occur in asthmatic persons. Desensitization follows the injection of minute amounts of the serum (0.1 c.c.), the full dose being given an hour later.

XIV. VINCENT'S ANGINA.

Definition.—An acute febrile, pseudomembranous inflammation of the tonsils, associated with *Bacillus fusiformis* and the spirochæta of Vincent, and characterized by a tendency to destructive ulceration of the tissues involved, enlargement of the lymphatic glands at the angle of the jaw, and an irregular, slow course.

This affection chiefly affects children and young adults. The diagnosis rests upon the bacteriological findings.

Symptoms.—The onset may be acute or subacute, with the symptoms of an ordinary angina or simple membranous sore throat. The constitutional symptoms are often severe. The tonsils are usually at first affected and, in some cases, the local manifestations of the disease are confined to those organs. More commonly the uvula and half-arches are also involved and the ulceration may extend to the pharynx and even to the gums. The exudate is of soft consistency, usually of a greenish or grayish-yellow color, and readily detached, leaving a slightly depressed bleeding ulcer with irregular ragged borders. The process is slowly progressive, not readily yielding to treatment, and may result in extensive destruction of the parts involved, especially the uvula and soft palate.

Fig. 241. — Fusiform bacilli and spirilla in Vincent's angina.

Diagnosis.—This form of membranous angina may be recognized by its tardy progress, the destructive tendency of the ulcerative process, and the presence of Bacillus fusiformis and the spirochæta of Vincent. The differential diagnosis from diphtheria turns upon the above anatomical and clinical characters and the absence of the Bacillus diphtheriæ from the exudate. In fact, diphtheria can usually be excluded when the associated fusiform organisms and spirochætæ are present. As a rule, the direct diagnosis depends upon the laboratory findings. Mucous patches may resemble this form of angina or it may develop upon syphilitic lesions.

Prognosis.—Recovery occurs as a rule after a duration varying from four or five days to several weeks. In a recent case in the Pennsylvania Hospital, Vincent's angina was the terminal event in aplastic anæmia in a young adult male.

XV. CROUPOUS PNEUMONIA.

Fibrinous Pneumonia; Lung Fever; Lobar Pneumonia; Pleuropneumonia.

Definition.—An acute infectious disease due to the Diplococcus pneumoniæ of Fraenkel and Weichselbaum and characterized by pulmonary inflammation and fever, usually of abrupt onset, high range, and critical termination.

Etiology.—Predisposing Influences.—Pneumonia prevails alike in hot and cold countries. It is said to be more prevalent in the temperate climates.

Season plays an important part as a predisposing factor. The incidence of the disease is uniformly greater in winter and the early spring. March is the month of greatest liability. This is in accordance with the fact that exposure to cold and especially unusual exposure to cold and wet are very often soon followed by the initial chill. Steady, low temperatures are less dangerous. *Personal Factors.*—Croupous pneumonia may occur in the new-born and in early infancy. It is common till the sixth year. The liability then diminishes until the fifteenth year. From fifteen to forty-five is a period of special liability. After sixty the disease is very common and often constitutes the terminal event in the aged, both in chronic disease and when the previous health has been well preserved. In infancy and old age the incidence for the sexes is about equal; in the middle period

of life, when the mode of living is different, males are more frequently affected than females. There is no special racial predisposition. The negro bears the disease badly. Rich and poor are alike liable. No occupation is conspicuous as a predisposing factor. Overwork and sudden exposure constitute especial risks. Lumbermen and miners frequently suffer. The disease is very common in cities. Pneumonia attacks the robust and hearty and the debilitated and previously ill with impartial energy. The alcoholic is especially liable. The last illness of the chronic invalid is very often pneumonia. Pneumonia sometimes follows injuries, especially contusions of the chest. This may occur in the absence of the signs of injury to the lung. The term "contusion pneumonia" is used to describe this variety.

Immunity.—There is apparently no natural immunity, certainly no permanent acquired immunity. On the contrary, croupous pneumonia is conspicuous among the infections for its liability to recur. Subsequent attacks have been noted in from 15 to 50 per cent. of the cases. I have seen a woman who stated that the attack was the twelfth, and several persons in whom a number of annual attacks occurred.

Exciting Cause.—Diplococcus pneumoniæ of Fraenkel and Weichselbaum, Micrococcus lanceolatus, or pneumococcus. This organism is the sole cause of true acute croupous pneumonia. It is present in the expectoration and pulmonary exudate in enormous numbers. Upon examination of the lungs, when death has occurred in the stage of resolution, it is found only in small numbers or may be wholly absent—phagocytosis. It may be obtained in blood cultures during the attack in more than 75 per cent. of the cases. It occurs commonly in pairs, each member being lancet-shaped, occasionally in chains of four or six cocci. In preparations from the lesions or fluids of animals and man the pairs are enclosed in a capsule. In cultures the capsule is frequently absent. The pneumococcus occurs in many other diseases, especially bronchopneumonia, pleurisy, endocarditis and pericarditis, meningitis, peritonitis, in forms of arthritis, and in middle-ear disease.

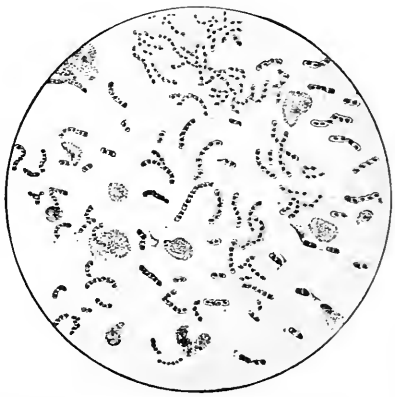


Fig. 242.—Spread of sputum showing pneumococci in pairs and in chains.

Much less commonly the infecting agent is Friedländer's pneumobacillus—*B. mucosus capsulatus.*

Cole and Dochez have shown that the pneumococci isolated from cases of pneumonia may be divided into four groups. The organisms belonging to each of the first two types are specific. An immune serum produced by the injection of a horse with pneumococci belonging to Type I has a specific protective action against all the members of Type I, but does not protect against the organisms of any of the other groups.

In like manner, an immune serum produced by the injection of a horse with a pneumococcus belonging to Type II is protective against all of the other members of this type, but has no effect against the members of any of the other groups. In Type III are included the organisms of the so-called *Pneumococcus mucosus* strain. It has not been possible to produce a protective serum against any member of this group. In Type IV are included all the cocci against which Serums I and II are not effective and which, from their other properties, cannot be classed in Type III. Animals may be immunized against any member of this group, and the resulting serum is protective against the strain used for immunization, but this serum is not effective against any other strain of this group or against the organisms of the other types. No constant group differences in cultural and morphologic characters have been discovered between the members of I and II and Type IV.

The pneumococci of Type I and Type II are identified by the agglutination reactions to their respective immune sera; those belonging to Type III by their cultural and pathogenic characters, and Type IV comprises the various types not referable to the above categories and is determined by exclusion.

In these studies white mice were used as laboratory animals and cultures and serum were injected at the same time into the peritoneal cavity.

Cole in a recent communication based upon 500 cases of pneumonia, in which the type was established, in the Hospital of the Rockefeller Institute, found "about one-third to be due to Type I pneumococci, one-third to Type II pneumococci, 10 to 15 per cent. to Type III pneumococci, and the remainder are due to infection with pneumococci belonging to the Type IV."

The determination of type as a basis of serum therapy has become very general in large city hospitals and the laboratories of state and municipal boards of health.

In this connection it is proper to state that the serum treatment properly carried out in cases due to Type I infection is clearly of value and has been followed by considerable reduction in the mortality.

Pneumococcus Septicæmia.—An acute general infection without localization in the lungs or serous membranes is occasionally encountered. This variety of pneumonia is analogous to the primary septicæmia due to general infection by Eberth's bacillus—typhoid septicæmia.

In its attenuated forms the pneumococcus is present in the mouth in a large proportion of healthy individuals. Its presence has been demonstrated in the dust of rooms. Various other organisms are associated with the pneumococcus in croupous pneumonia, as secondary or mixed infections. The most frequently encountered is the Streptococcus pyogenes. Less common are staphylococci, the bacillus of Friedländer, B. typhosus, B. diphtheriæ, and B. influenzæ. These bacteria are often the cause of bronchopneumonia, which in some instances closely resembles croupous pneumonia.

Pathological Anatomy.—The lesions in the inflamed lung undergo progressive changes, which, since the time of Laennec, have been described as the stage of engorgement, the stage of red hepatization, and the stage of gray hepatization. *Engorgement.*—The vesicular tissue is deep red, firmer than normal to the touch, and on section shows abundant blood and serum. It crepitates upon pressure, and excised pieces float upon water. The capillaries are distended and the air-vesicles contain blood-corpuscles and swollen detached alveolar cells. *Red Hepatization.*—The air-cells and terminal bronchi are occupied by the coagulated exudate, entangled in which are pneumococci, red blood-corpuscles, leucocytes, and alveolar epithelium. The affected portion of lung is solid and airless. It is enlarged and shows the oblique, parallel markings of the ribs. On section the surface is of a reddish-brown color and granular, an appearance produced by the protrusion of the fibrinous moulds in the vesicles. The terminal bronchi also contain branching fibrinous casts. The surface yields upon scraping a reddish, viscid serum. The hepatized lung is extremely friable. *Gray Hepatization.*—The lung tissue is now of a dirty gray color and more friable. The cut surface is more moist, has lost its granular appearance, and yields upon scraping a milky turbid fluid. The air again reaches the alveoli, from which the fibrin and red blood-cells have in great part disappeared, but in which are great numbers of leucocytes. The foregoing stages gradually merge into each other, and the process is not equally advanced in all parts of the lesion. The liquefaction of the exudate and resolution are the result of the action of proteolytic enzymes. The part of the lung occupied by the exudate varies from a small patch to the entire lung. Commonly a single lobe is involved. The uninvolved portions are usually congested and œdematous. The pleural surface opposite the exudate is always inflamed when the latter extends to the periphery of the lung. The bronchial glands are enlarged and sometimes softened.

The right heart is dilated. Pericarditis is not rare, especially in left-sided and double pneumonias. Endocarditis is common both in the simple and in the malignant form. Myocardial changes occur. The spleen shows moderate enlargement and, in the kidneys, parenchymatous swelling and the lesions of nephritis are frequently present. Meningitis is by no means rare and is often associated with malignant endocarditis. Diphtheroid colitis is rare. The liver is slightly enlarged and deeply congested. The distribution of the lesion is as follows: The right lung alone is involved in about 50 per cent. of the cases; the left alone in about 33 per cent.; both in less than 20 per cent. In double pneumonia the lower lobes are usually affected, or an entire lung with the lower lobe of the other. Much less frequently the lower lobe of one lung and the upper lobe of the other are involved—*crossed pneumonia.* Very rarely both upper lobes suffer.

Croupous pneumonia in the majority of the cases occurs as a sporadic disease. At the seasons of greatest prevalence it is the type of an endemic disease. House epidemics are by no means rare. In a family of five I have seen the mother and two children attacked in rapid succession, two

of the cases proving fatal. More extensive local epidemics occasionally occur in schools, prisons, and other institutions.

Symptoms.—In the well-developed cases of croupous pneumonia the attack runs a typical course and is self-limited. The period of incubation is of unknown duration. It is probably brief. Prodromes are unusual. When present they consist of slight catarrhal symptoms. The onset is abrupt, with a chill which is commonly severe and prolonged. The temperature rises rapidly to the fastigium—104°–105° F. (40°–40.6°C.). Headache, general pains, and the sensation of being very ill are followed in the course of a few hours by a severe stitch-like pain in the side, increased on full breathing, a short, dry cough, hurried respiration, and a full bounding pulse. Very significant is a short, expiratory grunt. The pain is often characteristic. The face is slightly cyanotic, with dusky, circumscribed flushing of one or both cheeks; the eyes are bright; the expression anxious; the nostrils dilate, and later patches of herpes, usually made up of many small vesicles, some of which contain blood, appear upon the lips and nose. The patient lies upon the affected side or flat upon his back. By the close of the second day there is scanty, viscid, blood-stained expectoration—*rusty sputum.* The physical signs of consolidation of the affected lung tissue are present—small mucous and crepitant râles followed by dulness and bronchial breathing. At the end of several days defervescence by crisis occurs with a remarkable amelioration of all the symptoms.

The following symptoms require special consideration: Fever.—The temperature rises rapidly, sometimes reaching the fastigium within twelve hours, usually within twenty-four hours. In childhood and old age the rise is more gradual, especially if there is no chill. Its course is subcontinuous and remarkably constant, the morning remissions and evening exacerbations in many cases not greatly exceeding the diurnal oscillations in health. Pseudocrises are not infrequent. They may occur at any period but are more common about the fifth or sixth day. They are usually single, but two or more sometimes occur. In the latter case the temperature range suggests an irregular intermittent. The defervescence in so-called classical cases is by crisis, which occurs between the third and twelfth days, very often upon an uneven day, and commonly upon the fifth or seventh day. A precritical rise of a degree or more is not very rare. The time occupied by the crisis varies from two to several hours. It is accompanied by an abundant sweat and usually occurs during a deep and prolonged sleep, from which the patient awakes weak but refreshed and comfortable. The fall almost always reaches subnormal ranges—96°–97° F. (35.5°–36° C.), and may be followed by a postcritical rise and subnormal oscillations for a few days. In some instances the crisis is interrupted by a rebound—*interrupted crisis*—or it may extend over twenty-four hours—*protracted crisis.* In delayed cases and in children the defervescence is often by lysis. In fatal cases of the so-called sthenic type there may be a preagonistic rise of temperature and in the asthenic cases an abrupt antemortem fall of several degrees. The crisis is sometimes attended by collapse symptoms. In the aged and in drunkards the temperature is much lower. Afebrile cases are encountered. Pain.—The pain

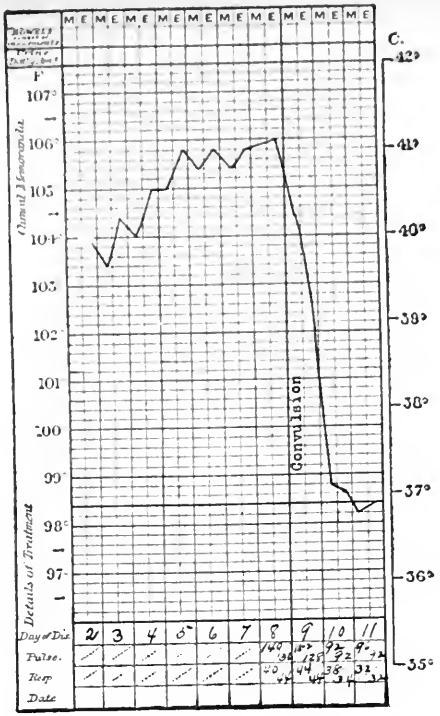


FIG. 243.—Pneumonia; acute nephritis. Convulsion at crisis. Recovery.

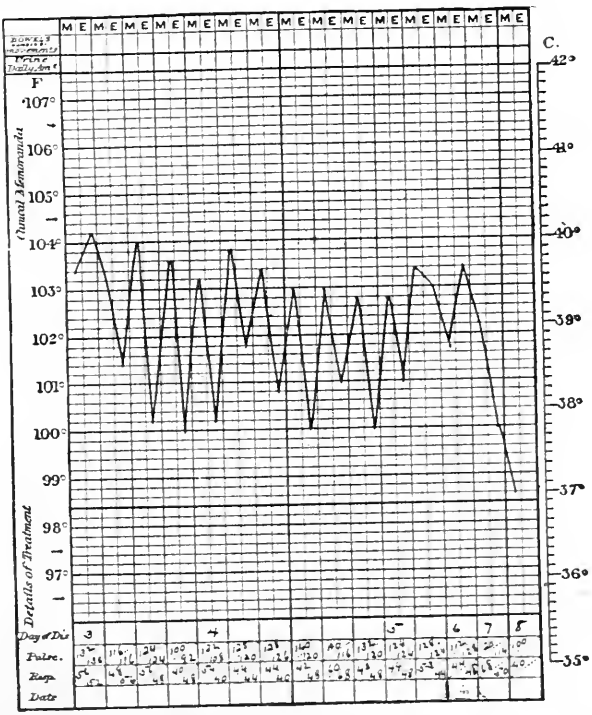


FIG. 244.—Croupous pneumonia. Recovery.

has all the characters of pleurisy. It is stitch-like and lancinating, usually severe, aggravated by deep breathing and cough, and referred to the region of the nipple or the infra-axillary region on the affected side. Occasionally it is referred to the epigastrium or the region of the appendix: an important point for the diagnostician. In these cases there is a diaphragmatic pleurisy. In apex pneumonia pain is less constant and less severe and in central lesions it is absent. Dyspnœa.—The respiration rate is increased in almost all cases, the masked pneumonias of drunkards and the aged and the terminal pneumonias of chronic diseases constituting

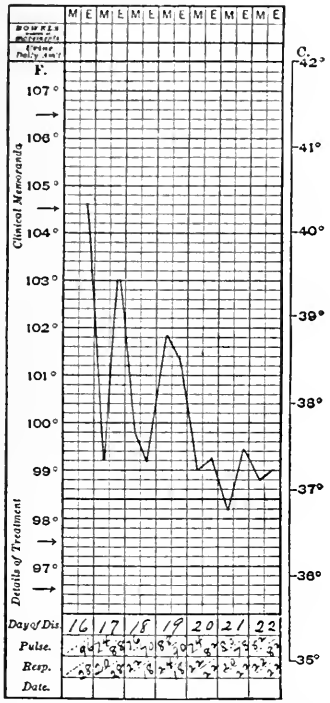


Fig. 245.—Croupous pneumonia. Prolonged febrile movement, terminating by lysis on 21st day of attack.

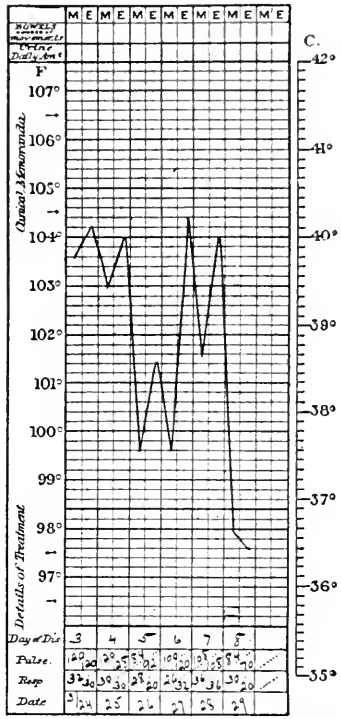


Fig. 246.—Pneumonia; pseudocrisis, 5th day; crisis 8th day.

the exceptions. The pulse-respiration ratio may be 2:1 or even 1.5:1. The respiration-frequency ranges from 30 to 50, and in children as high as 80 per minute in cases that recover; in fatal cases it is even more rapid. The breathing is shallow; the ancillary muscles are brought into play; there is, in many cases, respiratory distress associated with air hunger and a characteristic expiratory grunt or snort. Cough.—The early cough is due to pleural irritation and is short, hard, and dry. Later it becomes frequent, somewhat paroxysmal, and productive. It is attended with pain, often severe throughout the attack. About the time of the crisis, it becomes easier and is attended with free expectoration. Terminal pneumonias and those of the aged and drunkards are often without cough.

Absence of cough is also occasionally observed in the pneumonia of infancy. Slight cough after the crisis is often without significance; but severe paroxysmal cough may be a sign of pleural effusion. Sputum (see Part III, page 472, Vol. I). Pulse.—At the onset the pulse is small, but it soon becomes strong and full. It is seldom dicrotic. Later it becomes feeble and small. The frequency varies in favorable cases from 80 to 110. A very frequent pulse—130–140—is of unfavorable prognostic import, but less so in children than in adults. In feeble and aged persons the pulse is small and frequent from the onset. After the crisis the pulse remains frequent for a time but gradually returns to normal. The peripheral blood-pressure during the early days of the attack shows little or no change. Later there is often a progressive fall. A sudden drop may be the precursor of death. In the septic cases there is an early fall. Heart Sounds.—They are usually distinct and well-defined. The second pulmonary sound is accentuated. With engorgement of the right heart and incomplete systole of the right ventricle, the pulmonary second sound becomes progressively fainter. Great impairment of heart power shows itself in heart sounds of the fetal type. Sudden collapse may occur early in the disease, at the crisis, or during convalescence, and terminate in death. This may happen without warning in previously healthy persons with good hearts. Blood.—A decrease of the red corpuscles occurs at the time of the crisis but marked anæmia is not common. A leucocytosis varying from 10,000 to 50,000 per c. mm. is present in most cases throughout the attack. It disappears after the crisis. Its persistence may be the sign of a complication. It bears some ratio to the extent of the pulmonary exudate. In the toxic cases leucocytosis may be absent, and its absence in any case is an unfavorable sign. The blood-plaques and fibrin elements are increased. The eosinophile cells are decreased. Pneumococci can be isolated in some cases. Digestive System.—There is complete anorexia. The tongue, at first covered with a thick white fur, becomes red and glazed and in the severer cases dry and brown. Vomiting is frequent in children. Constipation is the rule. Meteorism is a troublesome condition in the graver cases. Fibrinous exudates have been observed in the mouth and nose and other mucous surfaces. Skin.—Herpes is very common—20–60 per cent. of the cases. It appears usually upon the lips at the border of the mucosa; less frequently upon the alæ nasi, infrequently upon the genitalia or anus, rarely upon the buttocks. Slight cyanosis may occur. Redness of the cheeks, and especially of the cheek upon the affected side, is very common. A general erythema is encountered in rare cases. As in all grave infections petechiæ may occur. Sweating is not common during the course of the attack, but is profuse at the crisis. Nervous System.—The symptoms referable to the nervous system are not peculiar to pneumonia but are, in certain cases, of great assistance in the diagnosis. In infants and young children convulsions may take the place of the initial chill. Headache is frequent and often severe. Insomnia is a troublesome symptom, often followed by delirium. The latter may be mild and wandering, becoming progressively more severe, even increasing after the crisis. In the intervals there is marked mental confusion. In a group of cases in children the symptoms suggest meningitis, and the actual condition is very

often overlooked. There are cases in which the onset is marked by furibund mania. In alcoholic cases the nervous phenomena closely simulate delirium tremens. Finally there are cases characterized from the onset by dulness and stupor, with no chill and but little fever, in which pulmonary symptoms are nearly or quite absent, but grave depression and wandering delirium constitute the only manifestations of profound toxæmia. The true character of such cases can only be determined by a systematic routine examination of the chest. Apex pneumonia is more frequently attended by severe nervous symptoms. The attack may be followed by postfebrile delusional insanity, which as a rule terminates in recovery. URINE.—The secretion has the usual characters of fever-urine. Toxic albuminuria is common. Later the signs of an acute nephritis may be present. Urea and uric acid, diminished during the attack, are greatly increased upon the occurrence of crisis. The chlorides are diminished or absent.

Physical Examination.—INSPECTION.—The attitude is variable. In lung lesions of moderate extent the patient lies upon his back or upon the affected side; in pneumonia of an entire lung, or double pneumonia, he usually prefers to be supported by pillows. The respiratory movement of the affected side is diminished. In basal pneumonia there may be increased movement over the upper lobe. The increased compensatory excursus on the sound side is often very conspicuous. The frequent breathing, the action of the auxiliary muscles of respiration, and the sudden muscular relaxation in expiration are to be noted. Orthopnœa may be present in severe cases. The affected side may look larger, but the increase upon actual measurement is trifling.

The difference in the expansion of the two sides is very evident upon palpation. The vocal fremitus is greatly increased over the lesion. It may be diminished or absent if the exudate extends into the middle-sized bronchi or a plug of tenacious mucus occludes a tube of some size.

PERCUSSION.—During the stage of engorgement the resonance is of higher pitch and vesiculotympanitic—*Skodaic resonance*. After hepatization has occurred percussion yields dulness, which varies from partial impairment of resonance with the tympanitic quality, to almost complete loss of resonance. Flatness is only present in massive pneumonia when the fibrinous exudate extends some distance into the larger bronchi. Beyond the borders of the lesion percussion often yields Skodaic resonance. As resolution takes place the dulness becomes less marked; the quality becomes vesiculotympanitic and by degrees the normal pulmonary resonance is restored. A certain elevation of pitch and faint tympanitic quality may persist for several weeks. Wintrich's phenomenon is sometimes present in apex pneumonia. In rare cases the percussion sound has an amphoric quality and suggests a cavity. In central pneumonia the symptoms may be well marked but percussion may fail to indicate the site of the exudate until it reaches the periphery of the lung, sometimes a period of several days.

AUSCULTATION. — In the stage of engorgement faint respiratory sounds. The tidal air is not only decreased in volume, it also ebbs and flows with diminished force. Then follow crepitant râles, heard only at

the end of inspiration—*crepitus indux*. In the stage of red hepatization, when dulness appears, the respiration becomes bronchial, at first soft and low-pitched, and more distinct upon expiration. Fully developed it is high-pitched, heard alike upon inspiration and expiration, with an interval of silence between the inspiratory and the expiratory sound and often, especially in the young, having a loud, snoring quality. In massive pneumonias in which the exudate fills the bronchi bronchial breathing is absent. Upon resolution small mucous and crepitant râles are again heard—*crepitus redux*—and are sometimes followed by larger bronchial râles which disappear as convalescence advances; more frequently by nearly or quite normal vesicular breathing. In central pneumonias the auscultatory like the percussion signs may be absent for a time. The variety of bronchophony known as ægophony is sometimes present, but it is a sign of trifling importance.

After the diagnosis is fully established it is not desirable to make frequent examinations of the chest. They are exhausting to the patient, especially as the crisis draws near, and in the absence of some special indication in the symptoms or general condition should not be repeated oftener than once in three or four days. In the necessary movements the patient must be carefully assisted and make as little effort on his own part as possible.

Complications and Sequels.—These are not many. Pleurisy.—Inflammation of the pleura corresponding to the exudate is always present when the latter extends to the periphery of the lung. It is usually fibrinous and cannot then be regarded as a complication. When serofibrinous, the effusion usually contains coarse fibrin flakes and there is much soft fibrinous deposit. It is often abundant. Even a moderate effusion coming on during the stage of hepatization may cause urgent pressure symptoms. A rare complication is pleural effusion upon the opposite side.

Metapneumonic Empyema. — This complication is not altogether infrequent. Cases are sometimes regarded as instances of delayed resolution. The pneumococcus is usually present early; the streptococcus afterwards. The signs of pleural effusion may appear during the attack or after the crisis. In the former case there may be pressure symptoms, as dyspnœa, cardiac embarrassment, and sensations of tightness, together with persistence of the fever. In the latter, the temperature rises and becomes remittent or intermittent and there are irregular profuse sweats, marked anæmia, leucocytosis, and not rarely paroxysmal cough. The diagnosis of small encapsulated and interlobar empyemata may often be made with confidence in cases in which their precise location remains obscure.

Pericarditis. — This complication is comparatively infrequent — 5 per cent. It occurs chiefly in left-sided or double pneumonias. The exudate is usually fibrinous. Precordial pain may be overlooked in connection with the pleurisy. A friction sound may be obscured by bronchial râles. The effusion may be serofibrinous or purulent.

Endocarditis.—Primary endocarditis may occur, or a fresh attack supervene in chronic valvular disease. The malignant form is occasionally associated with meningitis. The signs are not constant. Of diagnostic importance are murmurs which change their quality or point of maximum

intensity, irregular fever with chills and sweating, and signs of embolism. There are cases discovered post mortem in which no murmur has been recognized during life.

Thrombosis.—This condition may occur during convalescence. The femoral vein is commonly affected. Embolism of the larger arteries is very rare. Aphasia is also rare. It may occur with or without hemiplegia.

Meningitis is a rare complication occurring during the course of the attack or after the crisis. It has been observed more frequently during epidemics of cerebrospinal fever. It constitutes a most serious complication. The pneumococcus has been found in the exudate. Very rare

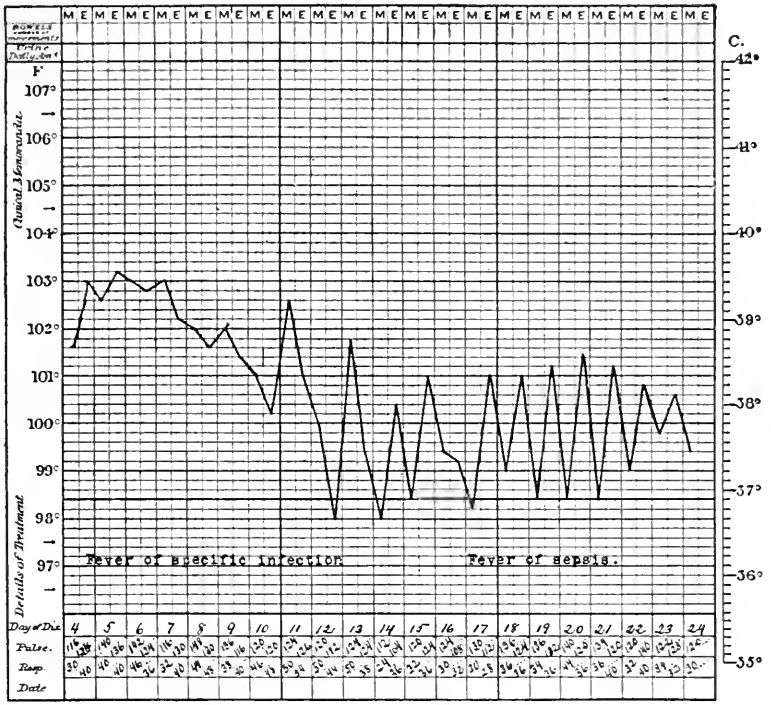


Fig. 247.—Pneumonia followed by empyema.

indeed are multiple neuritis, myelitis, and an ascending paralysis presenting the characters of Landry's paralysis. Postinfective insanity is rare. In individuals predisposed to neurasthenia an attack of pneumonia may precipitate the outbreak.

Abdominal Complications.—Croupous gastritis and colitis and acute dilatation of the stomach are rare. Appendicitis may occur as an intercurrent affection. Epigastric pain, which is especially common in children, is usually due to diaphragmatic pleurisy. It may simulate peritonitis—a fact of importance in diagnosis, since in rare instances inflammation of the upper peritoneum by extension from the pleuræ has been observed. The abdominal pain, if localized and attended with shock, may suggest acute hemorrhagic pancreatitis.

Jaundice.—Mild jaundice is not uncommon. It develops early and has no bearing upon the prognosis. A deep obstructive jaundice may occur.

Meteorism.—Abdominal distention is a common and troublesome symptom in the graver cases. It is the manifestation of the action of toxins upon the nerve supply of the walls of the gut. By mechanically interfering with the action of the diaphragm it adds to the embarrassment of the respiration.

Other Complications.—Parotid bubo occasionally occurs, especially in connection with endocarditis of malignant type. Middle-ear disease is common in children, and polyarthritis resembling that of rheumatic fever, sometimes clearly septic in character, may occur during the course of the attack or during the convalescence.

Relapse is a rare event. It is important not to mistake the fever of delayed resolution or of empyema for relapse. An initial chill, high fever, cough, rusty sputum, and critical defervescence would justify the diagnosis of relapse.

Convalescence is commonly rapid. Resolution does not immediately occur. Impaired resonance with the vesiculotympanitic quality and feeble vesiculobronchial respiration may persist for a fortnight or longer. Fever and a leucocytosis after the crisis suggest delayed resolution or empyema. A systematic physical examination and the use of the aspirator needle may become necessary. Persistent dulness is often due to a greatly thickened pleura.

Anatomically the terminations of croupous pneumonia are:

1. Resolution. — The exudate undergoes liquefaction and resorption. Only in small part is it expectorated. Complete restoration of the lung gradually occurs—*restitutio ad integrum.*

2. Delayed Resolution. — Resolution is more commonly delayed in pneumonias of the aged and in debilitated subjects; it may be delayed also in basal lesions and in previously robust persons. The crisis is followed by an apparent convalescence, but the signs of local consolidation continue. From three to five or six weeks elapse before dulness and bronchial breathing wholly disappear. Complete recovery ultimately follows. In a second group of cases the defervescence is by lysis, with irregular, recurrent fever, sweating, rapid pulse, slight cough, usually little or no expectoration, and impaired nutrition. The condition simulates pulmonary tuberculosis. In the course of four or five weeks, sometimes not until two months or more have elapsed, complete resolution takes place and the patient regains his health.

3. Abscess.—Local infection with pyogenic organisms. The abscess cavities are multiple and small or they may coalesce and form an extensive depot. Cough is paroxysmal and accompanied by an abundant purulent expectoration containing elastic fibres, sometimes cholesterin crystals and hæmatoidin crystals. The onset of the condition is commonly attended with increased fever of hectic type and other signs of sepsis. The differential diagnosis between a circumscribed empyema with bronchopulmonary fistula and pulmonary abscess rests largely upon the character of the sputum.

4. Gangrene.—Infection with saprophytic bacteria. The condition may occur independently of or in connection with abscess. The condition occurs mostly in persons debilitated by previous bad health. It

manifests itself clinically by septic phenomena and extreme prostration and by a disgusting, penetrating fetor of the sputum and breath.

5. Fibroid Induration.—Defervescence either by crisis or lysis may occur, but the signs of consolidation—dulness, increased vocal fremitus, bronchial respiration—continue unchanged. Occasional râles are heard. Cough may be troublesome, but expectoration is scanty. Resolution does not take place gradually as is usual, but the signs become more marked with contraction of the opposite side and the gradual development of chronic interstitial pneumonia—fibroid phthisis. In other cases the fever returns, the expectoration increases, and the course of the case is that of a rapid pulmonary consumption—phthisis florida.

Clinical Varieties.—The clinical picture of croupous pneumonia is made up of two essentially different groups of symptoms, namely, those due to the local pulmonary lesion and those due to the toxæmia. As these vary greatly, it is evident that the individual cases, while conforming to a type, will present differences which are determined among other factors by the relative preponderance of one or the other of these two symptom-groups. Complete consolidation of a lung may, on the one hand, be accompanied by but trifling evidences of general infection, while, on the other hand, severe, even fatal, toxæmia occasionally occurs in cases in which the pulmonary lesion is limited in extent. The other factors are: 1. Local Variations.—(a) *Apex pneumonia* is more common in children. It is frequently associated with marked cerebral symptoms and may simulate meningitis. In the absence of cough and sputum the pulmonary lesion is often overlooked. Pneumonia of the apex in adults may be accompanied by grave constitutional symptoms. (b) *Double pneumonia* is attended with the additional dangers incident to progressive diminution of the respiratory surface. The second lung is usually involved a day or two after the first and to a less extent. The extension of the process is not attended by a chill. (c) *Central Pneumonia.*—The exudate may remain circumscribed about the root of the lung or in the substance of a lobe and not reach the periphery for three or four days, when the physical signs may be for the first time detected. Meanwhile characteristic symptoms—chill, fever, cough, and rusty sputum—are present, but pain does not occur until the inflammation reaches the pleura. (d) *Pneumonia in Emphysematous Persons.*—The symptoms are distinctive, but, owing to the diminished vascular supply and the dilatation of the vesicles, there is not a sufficient quantity of fibrinous exudate to give rise to the signs of consolidation. Several days may elapse before the site of the lesion can be detected. (e) *Massive Pneumonia.*—A rare form in which the fibrinous exudate fills the bronchi. A lobe or the entire lung may be involved. The affected portion is converted into a completely airless mass. The percussion sound is not dull but flat. Upon auscultation neither râles nor bronchial respiration are heard, and vocal fremitus is absent. The signs closely simulate pleural effusion, but adjacent organs are not displaced. (f) *Migratory Pneumonia.*—The inflammation creeps about, involving in succession one lobe after another, resolution, not always complete, taking place in turn as new areas are affected. The migrations are not attended by chills; fever continues and the course of the disease is much protracted.

2. The Intensity of the Process.—(a) *Larval or Rudimentary Pneumonia.*—Mild cases occasionally occur. The symptoms consist of slight chill, moderate rise of temperature, and cough. The pulmonary signs are obscure. The expectoration is slight in amount and not usually rusty. Herpes is common. The attack comes to an end in the course of two or three days. Its true nature is often overlooked. (b) *Abortive Pneumonia.*—The attack begins with a severe chill. The fever is high. Pleural pains, cough, rusty sputum, herpes, and characteristic physical signs enter into a symptom-complex which is complete. In the course of the second or upon the third day the temperature falls by crisis and the patient enters upon convalescence. (c) *Intense Pneumonia; Sthenic Pneumonia.*—The adjectives sthenic and asthenic have to a great extent passed out of use, but almost every day one sees in a large hospital service cases of pneumonia of great severity but in strong contrast as regards the character of the symptoms. Those terms serve a useful purpose in this connection. Intense pneumonia occurs in middle life, in individuals previously in good health, usually males, large, deep-chested, hard workers in the open air. The initial chill is severe and prolonged, the temperature high, the pulse bounding, the face flushed, chest pain very intense, the breathing hurried, cough frequent, sputum hemorrhagic, the delirium active, even maniacal, and the signs indicative of an extensive pulmonary inflammation. These cases are attended with especial danger to life. (d) *Toxic, Asthenic or So-called Typhoid Pneumonia.*—This variety, equally severe and even more dangerous, is in the strongest contrast to the variety just described. The ordinary symptoms of pneumonia may be absent and the pulmonary lesions limited. The symptoms are those of profound toxæmia. Early prostration, delirium, jaundice, meteorism, and diarrhœa are present. The hands and lips tremble and stupor alternates with wandering delirium. The condition is one of pneumococcus septicæmia or mixed pneumococcus and streptococcus infection.

3. Individual Tendencies.—(a) *Age.*—In the new-born croupous pneumonia is extremely rare. It is common in infants and young children. Convulsions replace the initial chill. The apex is not rarely involved. Cough is slight and suppressed or absent altogether. There is no expectoration. Excitement, jactitation, boring of the head into the pillows, and high fever followed by stupor and convulsions suggest meningitis. The apex pneumonia is often wholly overlooked. Pneumonia in the aged usually develops insidiously without a distinct chill. There is little cough and expectoration. Fever is moderate and irregular and the physical signs not well defined. Great depression, inability to take nourishment, mild delirium, and a tendency to stupor are present. (b) *Sex.*—In women at the middle period of life pneumonia tends to assume the toxic form, in men the sthenic form; in infancy and old age the course of the disease is the same in males and females. (c) *Pneumonia in Alcoholic Subjects.*—Two forms are to be considered—pneumonia in steady drinkers and pneumonia during debauch. In the first the early symptoms do not differ from those of ordinary pneumonia. Delirium with tremor soon develops, vomiting is troublesome, the circulation fails, sleeplessness is uncontrollable, and the signs of nephritis with uræmic phenomena are common. In the second

the onset is insidious, the temperature but slightly raised, cough, expectoration, and sputum trifling or wholly absent, and the clinical picture that of delirium tremens. Only by a systematic physical examination can the condition be recognized. (d) *Pneumonia in Chronic Diseases; Terminal Pneumonia.*—The terminal event in many chronic diseases, especially pulmonary tuberculosis, valvular and myocardial disease of the heart, arteriosclerosis, nephritis, diabetes, cancer, and diseases of the spinal cord, is croupous pneumonia. The development of this intercurrent disease is very frequently overlooked, partly because it is very insidious and presents none of its ordinary symptoms, and partly because the patient has reached a point in the progress of the primary affection in which a proper physical examination can no longer be made. The diagnosis is frequently made in the post-mortem room. The *intercurrent pneumonias of the acute infections*, as enteric fever, diphtheria, and influenza, are not as a rule due to the pneumococcus, but to the specific organism of the primary disease in association with secondary invading bacteria—Streptococcus pyogenes, staphylococcus, or the colon bacillus.

4. Varieties due to Differences in the Determining Causes.—(a) *Contusion Pneumonia.*—Contusion of the chest, or violent bodily shock without direct injury to the lung, may be followed in the course of a day or two by the onset of a well-characterized croupous pneumonia. (b) *Postoperative Pneumonia.*—The cases probably do not all belong to the same group. True croupous pneumonia is much less common than bronchopneumonia, which may be diffuse or pseudolobar. Croupous pneumonia may occur after operations of various kinds, irrespective of the anæsthetic employed. Bronchopneumonia is common after operations upon the mouth and throat. (c) *Anæsthesia Pneumonia.*—This variety is almost always bronchopneumonia. In many instances the lesions are so massed as to constitute a pseudolobar pneumonia. The symptoms develop in the course of the first or second day after the operation, much more commonly when ether has been administered and the mouth, throat, or abdomen has been operated upon. It is probably an aspiration pneumonia.

Diagnosis.—Direct.—In well-developed cases of primary croupous pneumonia the diagnosis is an easy matter and errors are not often made. The mistakes in diagnosis occur mostly in the aberrant and intercurrent forms in which the disease is latent and the symptoms masked, and are the result of neglect to carefully and systematically examine every patient, and especially chronic cases, upon the appearance of fresh local or constitutional symptoms, however trifling they may appear. In certain cases the general symptoms are indeterminate but the local phenomena decisive, in others the physical signs are obscure, but chill, fever, cough, and sputum are characteristic. In either of these conditions the diagnosis is clear; still more clear is it when both symptoms and signs are present and well defined.

Differential Diagnosis.—1. *Acute Pneumonic Phthisis* (see p. 199). 2. *Hemorrhagic Infarct.*—There are circumscribed dulness and bloody sputum. The chill of pneumonia does not occur; there may be complete absence of fever; the blood is less thoroughly admixed with the sputum, and finally a condition capable of giving rise to embolism may be dis-

covered. 3. *Pulmonary Œdema.*—The sputum is bloody, but it is also thin and frothy, a condition only exceptional in pneumonia. Dulness is not common and when present involves both bases posteriorly and is far less strictly delimited than in pneumonia. Both conditions may be present. Collateral œdema—fluxion œdema—is common in pneumonia and an inflammatory œdema may develop at the borders of the lesion. 4. *Bronchopneumonia.*—Massed lesions of considerable extent or involving a lobe—pseudolobar pneumonias—are misleading. Croupous pneumonia generally occurs as an acute process, attacking persons in previous good health, or as an intercurrent specific disease in various chronic affections, whereas bronchopneumonia is mostly an affection secondary to some acute specific fever, as measles or other condition in which the aspiration of infectious matter from the mouth or throat takes place. Croupous pneumonia is furthermore an acute, short, well-characterized disease, beginning abruptly with a chill and terminating by crisis, while bronchopneumonia comes on gradually or abruptly with temperature rise but without chill, continues indefinitely, and terminates in favorable cases by lysis. In the cases in which the diagnosis is uncertain, sputum is often absent, but, as a rule, to which, however, there are exceptions, when present it is rusty and viscid in croupous pneumonia and mucopurulent in bronchopneumonia. 5. *Pleural Effusion.*—This question of diagnosis is of daily occurrence at the bedside and demands special consideration (see p. 504). 6. *Meningitis.*—Doubts arise in some cases of apex pneumonia, especially in children. A knowledge that the pulmonary lesions are often masked will remind the practitioner that in every case in which meningeal symptoms are present the lungs and heart are to be particularly examined. 7. *Enteric Fever.*—There are two principal sources of error. Patients suffering with toxic pneumonia or the asthenic form of the disease present septic symptoms identical with those of enteric fever with mixed infection. Clinically, in the absence of a satisfactory anamnesis the diagnosis is often obscure. A positive agglutination with the Widal test is mostly conclusive. Exceptionally the patient may have passed through enteric fever some weeks or months before. To this condition the term *typhoid pneumonia* is frequently applied. This unfortunate term is also used to designate cases of enteric fever in which bronchopneumonia—inhalation pneumonia—has arisen as an intercurrent condition. Much less common are cases of enteric fever which begin with the symptoms and soon develop the signs of pneumonia—*pneumotyphus*. The diagnosis cannot be made with precision until the eruption and splenic tumor appear or a positive result follows the agglutination test.

Prognosis.—Croupous pneumonia, taking all cases together, is an extremely fatal disease. The statistics are unreliable. When the facts in the natural history of this disease are considered it appears probable that the mortality, according to hospital statistics, is too high; according to the impressions of physicians in private practice, too low. Especially misleading are the figures collected to support the efficacy of certain methods of treatment. It is necessary to be explicit. The clinical varieties are to be considered. Apex pneumonia is more liable to be associated with nervous symptoms and grave toxæmia. Double pneumonia is attended with increasing circumscription of the respiratory surface and stress upon the

right heart, as well as by a more intense toxæmia. In the migratory form there is increased danger from the prolongation of the active disease. As regards the intensity of the process, the powers of resistance of the individual play an important part. On the one hand robust and previously healthy persons, free from alcoholism, perish in a few days, while individuals of feeble constitution recover from an apparently hopeless attack. The toxic cases are mostly fatal. Nevertheless the better the previous health, the more favorable the outlook. That wholly unknown influence called by the older writers the epidemic constitution is far from being unimportant. The mortality varies in different years in the same locality between 5 and 30 per cent., and without discoverable cause there are alternating series of favorable and unfavorable cases. The mortality in house epidemics and institutions is high. Negroes in the United States show an increased mortality. The death-rate is distinctly higher in the southern than in the northern states of our country. In high altitudes the prognosis is extremely unfavorable. The question of diagnosis has a distinct bearing upon the statistics. The pneumonias of infancy and old age, secondary pneumonias, and terminal pneumonias are very often not recognized. In infants bronchopneumonia is frequently mistaken for croupous pneumonia or the latter for meningitis. In the aged, pneumonia frequently causes death without characteristic or even suggestive symptoms. Insidiously developing intercurrent pneumonias may be wholly overlooked and the fatal issue ascribed to the primary disease. Certainly this is true of terminal pneumonias—a fact which accounts for the discrepancy in the death-rate from pneumonia as reported from the wards and upon the protocols of the post-mortem room. It is easy to overlook pneumonia in a patient dying in the ward of a chronic disease; impossible to do so upon the autopsy table.

Statistics, to be of value, especially to be of value in determining the relative efficacy of different plans of treatment, must be based upon large numbers of cases analyzed with reference to all the factors which influence the result of the attack in individual instances. In hospitals the mortality ranges from 20 to 40 per cent. In the Pennsylvania Hospital, of 943 cases entered as pneumonia in seven years, 198 or 21 per cent. died. In the German Hospital, of 407 cases treated during ten years 108 died, a mortality of 26.5 per cent. The mortality in private practice varies according to different observers from 3 or 4 to 20 per cent. The series of cases in private practice are usually too small to be of statistical value.

Among the circumstances which bear upon the prognosis in individual cases are the following: Under one year the death-rate is much higher than between two and twelve. Adolescents and healthy young adults bear pneumonia well. The death-rate is very low among recruits and young soldiers, picked men living a regular life in well-constructed barracks. After sixty, 75 per cent. die; yet remarkable recoveries occur. I have now under observation a lady aged 99 who has twice had well-characterized croupous pneumonia since her eightieth year. Women bear pneumonia comparatively badly. When it occurs during pregnancy there is danger of abortion or premature labor, but the danger is not so great as it was at one time thought to be. In those previously ill with chronic

disease, the obese, and especially in those habitually given to drink, pneumonia is especially dangerous. In such cases also astonishing recoveries occasionally take place. The outlook is also grave in gouty persons and those suffering from emphysema. Complications add greatly to the gravity of the cases. Pneumococcus meningitis may be regarded as a fatal disease; endocarditis is usually of the malignant type; septic phenomena, whether due to the toxæmia of the primary infection or to secondary infection, are ominous. A low leucocyte count is unfavorable.

Cole's statistics (1917) "indicate that the cases due to Type I and Type II are of average severity, the mortality being from 25 to 30 per cent.; those due to Type III are severe, one-half or more of the patients dying, while the cases due to organisms of Group IV are milder and the mortality is usually not more than 10 to 15 per cent."

Death is commonly caused by the action of the toxins upon the vasomotor centres with progressive lowering of the blood-pressure; sometimes by acidosis. In many cases over-distention of the right heart is at fault. Sudden œdema of the lungs frequently precedes the fatal event.

XVI. CEREBROSPINAL FEVER.

Epidemic Cerebrospinal Meningitis.

Definition.—An acute, infectious, epidemic disease caused by the Diplococcus intracellularis meningitidis, characterized clinically by sudden onset, with headache, vomiting, and painful contraction of the muscles of the back of the neck, irregular fever, profound nervous symptoms, rapid course, and high death-rate; anatomically, by inflammation of the meninges of the brain and cord.

Etiology.—Predisposing Influences.—Climate appears to have little influence as a predisposing factor. Outbreaks are more common and extensive in the winter and spring than in the warm seasons of the year. Densely populated cities and sparsely settled agricultural regions are alike subject to its prevalence. Damp, overcrowded, and unclean habitations favor its spread, and persons living on the ground floor are especially apt to suffer. Individuals of all occupations and professions are liable to this disease. Military life involves a special liability. Among adults the proportion of males attacked is greater than that of females. Among children the number of males and females is about equal.

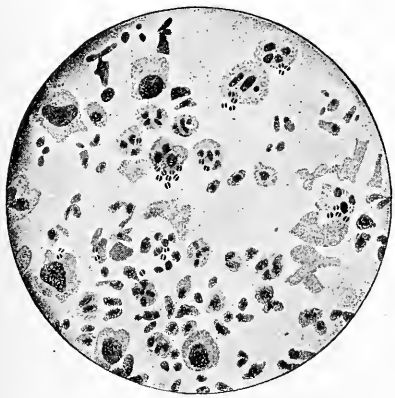


Fig. 248.—Spread of meningococcal exudate showing intracellular meningococci.

Cerebrospinal fever is especially a disease of children and young adults. After 40 it is uncommon, though the diagnosis has been verified post mortem in individuals over 70.

Exciting Cause.—Diplococcus intracellularis meningitidis; meningo-

coccus. This organism is found in the fluid obtained by lumbar puncture and in the meningeal exudate. The cerebrospinal fluid is usually more or less turbid, sometimes very turbid, especially early in the course of the attack. While turbidity of the spinal fluid is of diagnostic importance, its limpidity does not constitute a negative sign, and in either case cultures are necessary to the diagnosis. The diplococcus is almost constantly confined to the interior of the polynuclear leucocytes. It is invariably present in the lesions of the disease. Mixed infections are not uncommon.

Cerebrospinal fever is not contagious in the sense in which we use the term in speaking of smallpox, scarlet fever, and typhus. The definite micro-organism which causes it is, in the majority of instances, confined to the meninges of the brain and cord, with little or no opportunity of transmission to other individuals. In cases in which there are lesions in the lungs, ears, and nose, however, infection of neighboring objects or persons may readily take place. "The presence of sporadic cases is of importance in the occurrence of epidemics. The Diplococcus intracellularis is an organism of feeble vitality; it dies out easily on exposure to drying and light and is incapable of a saprophytic existence. In the absence of intervening infections, it would be impossible for the period of epidemics to be bridged over. Not only this, but there is evidence that this organism may even live as an inhabitant on the normal mucous membrane." (Councilman.)

In most cases the attack is followed by lasting immunity.

Symptoms of the Ordinary Forms. — Cerebrospinal fever presents a great diversity of symptoms in different cases. No other acute disease appears in such various guises. Stillé has well called it a "chameleon-like disorder." The period of incubation is unknown. Prodromes are rare. When present they consist of headache, dragging muscular pains, vertigo, and a sense of fatigue. The onset of the attack is usually abrupt. It is marked by a chill, agonizing headache, nausea, and vomiting. In some cases headache is not a conspicuous symptom. There is vertigo, and the patient acts like a drunken man. Herrick in a recent study of 208 cases at Camp Jackson lays especial stress upon the fact that the meningitis is preceded by the symptoms of generalized infection lasting from a few hours to two or three days. Dragging pains in the neck spread along the spine and into the extremities and are followed by motor symptoms which progressively develop. These consist of tetanoid stiffness of the spinal muscles, great pain on attempting to bend the head forward or to turn it from side to side and awkwardness and difficulty in movements of the extremities. Strabismus, inequality of the pupils, and palsies of the facial muscles are common. In the course of a little time opisthotonos develops, the head is drawn back, the spine curved, the forearms fixed on the arms and the legs on the thighs. Muscular cramps and spasmodic twitchings occur and in young children general convulsions. Hemiplegia has been frequently observed. Paraplegia may also occur. Paralysis may develop during the course of the attack and disappear shortly or persist for some time. The sensory symptoms consist of headache, which may be sharp, lancinating or boring, and is commonly referred to the back of the head;

sometimes it is felt as a constricting band; pain in the back of the neck and in the lumbar and epigastric region and general hyperæsthesia, most marked in the face and neck. Photophobia, intolerance of sounds, ringing in the ears, and vertigo, occur. The patient is restless and distressed. His face is seldom flushed, usually pale, and slightly cyanotic. In children there is great irritability. Delirium occurs early and may be active, even maniacal, or of a busy, wandering type. After a time it passes into somnolence or stupor, which may be still attended by more or less restlessness. In the worst cases stupor deepens to coma. The tongue is at first slightly covered with a whitish fur. In conditions of great depression it becomes dry and brown and sordes collect. Taste is lost and the patient refuses food; nevertheless the vomiting persists. Constipation is commonly present throughout the sickness. Toward the end of the attack diarrhœa and involuntary discharges may take place. Enlargement of the spleen may be made out. The fever is generally moderate, very irregular, and does not observe a typical course. There are frequent remissions. It may be slight or absent altogether. On the other hand, the temperature may reach 105° or 106° F. (40.5°–41.1° C.). It may abruptly rise before death. The fever, even when intense, may be of short duration. There is no constant relation between the intensity of the febrile movement and the severity of the other symptoms. Defervescence may take place without improvement in other respects, and severe nervous symptoms may persist for weeks after the temperature has fallen to normal or subnormal ranges. Many of the cases show a temperature range of irregularly remittent type. In the milder cases the temperature is sometimes distinctly intermittent. The subfebrile temperatures are sometimes broken by rapid and transient elevations. The pulse is also irregular. There is no constant correspondence between the pulse and temperature. It may be soft and weak, even slower than in health, and is often intermittent and arrhythmic. Abrupt changes in the force and frequency of the pulse are common. Change in the frequency from 80 to 100 has been observed in the course of a minute. The rhythm of the respiration may be disturbed and Cheyne-Stokes breathing may occur.

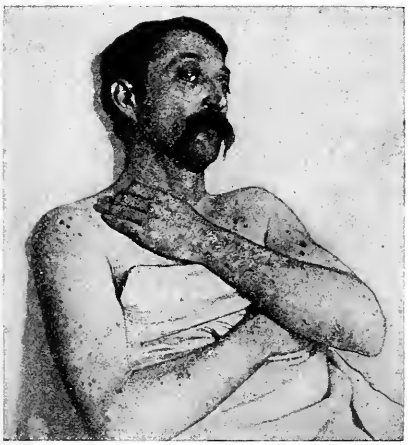


Fig. 249.—Petechial eruption; epidemic cerebrospinal meningitis.—Royer.

A polymorphonuclear leucocytosis is present throughout the disease, diminishing toward the end of the attack in cases which recover.

Lesions of the skin are common. To their prominence is due the old name of spotted fever. They vary greatly in different epidemics. In many cases they are absent altogether. They are often polymorphous. Herpes is far more common than any other eruption. It usually appears on the lips and nose, but may involve other parts of the face or body and may vary from a crop of a few fine vesicles to an abundant eruption of large vesicles.

A petechial rash resembling flea-bites is frequently observed, and in some instances extensive hemorrhagic areas develop in the skin. These lesions are distributed over the whole surface, but particularly about the knees and elbows. In some cases the rash is abundant and develops with great rapidity. Patches of erythema, dusky mottlings, and rose spots disappearing on pressure, like the rash of enteric fever, have been observed. Among the rarer cutaneous manifestations are urticaria, erythema nodosum, pemphigus, and gangrene. The urine is, as a rule, increased. It may be much increased even with high temperature. The reaction is usually acid. Albumin is frequently present. There is a special form of cerebrospinal fever characterized by symptoms of an acute nephritis and corresponding to the renal form of enteric fever. Glycosuria occasionally occurs, and in malignant cases hæmaturia has been observed. Retention of urine is common in the graver cases. Polyuria is frequent in children, and in some cases has persisted for years after convalescence.

The eye lesions are referable to three causes: First, neuritis, due to the involvement of the nerve in the exudate at the base without extension of the inflammatory process to either the orbit or the eye. This condition may affect the oculomotor and the optic nerve. Second, inflammation from the meninges may extend directly into the eye along the pia-arachnoid of the optic nerve, causing purulent choroido-iritis and in very rare instances suppuration in the orbit. Keratitis may arise in consequence of an extension of the inflammation from the iris and ciliary region. The third cause is neuritis of the fifth nerve, with loss of sensation and keratitis and purulent conjunctivitis. Symptoms relating to the auditory apparatus are common. The auditory nerve is swollen and surrounded by the exudate. Extensive degeneration of the nerve-fibres is frequently found, being most marked in the chronic cases. The abortive form of epidemic cerebrospinal meningitis is the cause of many cases of early acquired deafness. Deafness is frequently due to disease of the labyrinth. Otitis media and mastoid disease occur. The diplococci are found in the pus-cells. Coryza has been frequently observed in the course of the attack. Weigert first advanced the opinion that in meningitis the nose forms the portal of entry for the infectious organisms. It may be, however, that their presence is due to an extension from the brain and not to primary invasion. Epistaxis also occurs. The wasting in severe cases is rapid and extreme. An early, sudden, and great loss of strength is a frequent and prominent condition in this disease.

The symptoms may be divided into those due to the inflammatory lesions of the cerebrospinal organs and those due to a general infection. In the malignant cases both these groups of symptoms are of overwhelming severity. In the mild cases the nervous symptoms are predominant. The foregoing symptoms indicate the nature and severity of the disease in its ordinary form.

Anomalous Forms.—1. Malignant (*Meningitis Cerebrospinalis Epidemica Siderans*).—The patient is struck down without warning and speedily falls into a state of collapse. A violent chill is followed by cyanosis, coldness of the surface, profuse perspiration, intense headache which alternates with drowsiness, and brief delirium followed by unconsciousness. There may be contraction of the neck. Respiration is slow and labored;

the pulse rapid and feeble; the urine scanty and loaded with albumin. Purpuric blotches appear on the surface. Cases of this kind have occurred in many epidemics and with greatest frequency at the beginning of the outbreak. They may occur sporadically. Death may ensue in the course of a few hours. 2. Abortive (*Meningitis Cerebrospinalis Epidemica Abortiva*).—The onset of the attack is severe. In the course of a few days the symptoms subside and convalescence is rapid. 3. Mild (*Meningitis Cerebrospinalis Epidemica Ambulans*).—Patients complain of headache, stiffness in the neck and spine, and malaise. Vomiting occurs. Fever is, as a rule, absent. Cases of this kind can only be recognized in the light of a prevailing epidemic. 4. Intermittent (*Meningitis Cerebrospinalis Epidemica Intermittens*).—This form is common. Not only the fever, but other symptoms of the disease show extraordinary exacerbations and remissions, which may be repeated at intervals of twenty-four or forty-eight hours. These cases may be due to successive involvement of areas of the meninges or to fresh growths of the organisms. They rarely present the well-marked periodicity of the malarious diseases. 5. Chronic (*Meningitis Cerebro-*

Fig. 250.—Cerebrospinal fever; 53d day of the attack.—Pennsylvania Hospital.

spinalis Epidemica Chronica).—Cases of this form occur in all epidemics. The disease lasts, with numerous complications, remissions, and exacerbations, for several weeks or in some instances for five or six months. Emaciation is extreme. The symptoms may be due to the persistence of conditions left by the acute attack, such as chronic hydrocephalus or abscess of the brain, or general neuritis.

Complications and Sequels.—Among the complications and sequels are pleurisy, endocarditis, and pericarditis. Bronchial catarrh and deglutition pneumonia are very common. Croupous pneumonia has been common in some of the epidemics. It occurs more frequently at the close than at the beginning of an epidemic. Whether cases of pneumonia reported in connection with epidemic meningitis have been cases of true croupous pneumonia or cases of meningococcus pneumonia is uncertain. Arthritis, commonly slight, but in rare instances suppurative, has been noted. The wrist-joints are most commonly involved. Swelling of the parotid glands is an occasional accident of the disease. It may be slight or may run on to suppuration—*parotid bubo.* Intestinal catarrh may occur as a complication. Malarial and enteric fever, and measles, scarlet fever, and cholera have been encountered as intercurrent affections.

The convalescence is irregular and uncertain. After severe cases it is apt to be tardy. Relapses are not uncommon and are often fatal.

Among the more important sequels are prolonged debility and emaciation, palsies and various forms of paralysis, impairment of intelligence in consequence of chronic meningitis and chronic hydrocephalus, especially in children, and more or less complete deafness and loss of vision. General motor weakness and paralysis of individual cranial nerves or of the lower extremities may persist for a long time. They depend on lesions of the brain or spinal cord, or pressure exerted by extensive organized inflammatory exudate, or on peripheral neuritis.

Meningismus.—This term is used to indicate a group of symptoms suggestive of meningitis in cases in which recovery usually takes place rapidly and completely or in which after death the lesions of meningitis are not found. The fluid obtained by lumbar puncture is normal and free from organisms. It has been observed in various febrile infections, especially in children. The initial symptoms are frequently severe and the early differential diagnosis sometimes impossible.

Diagnosis.—Direct Diagnosis. —The recognition of cerebrospinal fever by ordinary clinical methods is a matter of difficulty in sporadic cases and at the beginning of outbreaks. The diagnosis of any form of meningitis is occasionally obscure. Sudden onset, chill, fever, vomiting, delirium, tremor, and painful rigidity of the back of the neck may occur in pneumonia, the malignant form of variola, typhus, and especially in the cerebrospinal form of enteric fever. Kernig's sign is found to be present in 80 to 90 per cent. of the cases of meningitis and only exceptionally present in other diseases. This test is often attended by evident pain on the part of the patient. Brudzinski's sign is common.

If meningitis be present there is usually no great difficulty in recognizing cerebrospinal fever during an epidemic. The ordinary and anomalous forms alike show a symptom-complex that in the course of a little time is distinctive. In sporadic and atypical cases lumbar puncture should be performed. If carried out early in the attack, at the time when the diagnosis is often as important as it is difficult, the result is commonly conclusive.

Fig. 251.—Kernig's sign in epidemic cerebrospinal meningitis.—Royer.

Lumbar Puncture (Quincke). —The operation is devoid of danger and can be performed without general anæsthesia. Freezing of the skin may be dispensed with, as it is as painful as the puncture and causes unnecessary delay. In children excitement may be avoided by a few whiffs of chloroform. Surgical antisepsis is to be strictly observed. Suitable pointed cannulas are sold in the shops. A small aspirator needle may be used. The instrument is introduced into the subarachnoid space between the fourth and fifth lumbar vertebræ. The

point of entrance may be determined by drawing a line connecting the highest points of the crest of the ilium posteriorly. This line passes over the spine of the fourth lumbar vertebra. The point of entrance is about one centimetre below and one centimetre to the right of the intersection of the transverse line and the median line. Some prefer the third lumbar interspace. The patient should lie upon the right side, the spine being strongly bowed, the thighs and knees flexed, and the left shoulder drawn forward. The thumb of the left hand being used as a guide, the needle is thrust with a rotary movement in an upward and inward direction to a depth varying, according to the age of the patient and the thickness of the tissues, from about two and a half centimetres in infants to between four and six centimetres in adults. The fluid runs drop by drop or in a stream, the normal pressure being about 120 mm. of water. In meningitis the pressure may reach 250–300 mm. Normal fluid is clear and limpid, but no conclusions can be reached without careful laboratory investigation, including, in doubtful cases, the inoculation of a guinea-pig.

If the patient has meningitis the fluid withdrawn is, as a rule, but not invariably, more or less cloudy; if cerebrospinal fever, it contains polymorphonuclear leucocytes with intracellular meningococci, and the Diplococcus intracellularis meningitidis—meningococcus—will be found on direct microscopic examination or in cultures. Positive conclusions can only be drawn from positive results. When the result is negative the operation must be repeated.

Differential Diagnosis.—1. *Pneumococcus Meningitis.*—This may occur alone or in connection with croupous pneumonia. The pulmonary lesion may be latent. Symptoms indicating extensive infection of the meninges, of the cord and spinal roots, and extension of the infective process along the cranial nerves are less marked or absent altogether. Contraction of the muscles of the neck may be absent, delirium and coma are present and occur early, and this form of meningitis is fatal, while cases of the epidemic form may recover.

2. *Streptococcus Meningitis.*—This form is secondary to infection elsewhere. Fracture of the skull, especially fracture of the base, local abscess formation, acute endocarditis, erysipelas of the face and scalp, otitis media with extension to the mastoid or the meninges are forms of primary infection. Opisthotonos is neither common nor well developed. The symptoms develop slowly and are often for a time obscure. The association of painful rigidity, intense headache, and vomiting is not conspicuous. Eye symptoms are common.

3. *Tuberculous Meningitis.*—This, perhaps the most familiar form of meningitis, is to be distinguished from cerebrospinal fever by a protracted period of prodromes, more gradual onset, slower course, slow and irregular pulse, great irregularity of the respiration, and the absence of eruption. Antecedent tuberculous disease, failure of health following measles or influenza, tuberculous glands, or a hereditary predisposition to tuberculous infection are found in the history of the patient. In children or during the prevalence of an epidemic of cerebrospinal fever and in cases in which the tuberculous process involves the spinal meninges (Hirsch), the diagnosis is far from easy. The result of spinal puncture is conclusive.

4. *The Cerebrospinal Form of Enteric Fever* (see p. 29).

5. *Scarlet Fever.*—In some instances the sudden onset, high febrile movement, vomiting, convulsions, and stupor suggest cerebrospinal fever as it occurs in children. The presence of the peculiar redness of the palatine half-arches, rapidly followed by general erythematous angina, are important. In the course of twenty-four or thirty-six hours the efflorescence will clear up any uncertainty.

6. *Typhus Fever.*—At one time cerebrospinal fever was confounded with typhus or regarded as a variety of that disease. To Stillé is due the credit of having finally settled every question of doubt concerning the identity of these two diseases in this country. They are in strong contrast in respect of their causes, symptoms, course, lesions, and sequels.

7. *Hysteria.*—Cases of cerebrospinal fever, occurring in nervous females at the close of epidemics or sporadically, have presented a delirium so peculiar and an array of symptoms so little characteristic that they have been looked upon as manifestations of hysteria. This error in diagnosis is no longer possible.

Prognosis.—The prognosis can never be made with certainty. Abortive and fulminant cases run a rapid course. Certain cases, which at the onset present the symptoms of cerebrospinal fever, recover after an illness of a few hours which terminates in free sweating. The malignant cases, on the other hand, prove fatal in a few hours or two or three days. Moderately severe cases may last one or two weeks or several months. The first week is the time of greatest danger. Symptoms rendering the prognosis unfavorable are intense excitement, early depression, persistent vomiting, irregular respiration, and convulsions alternating with coma. The average mortality is about 40 per cent. It varies in different epidemics from 20 to 75 per cent. Relapses are not infrequent and are often fatal. Efficient treatment by antimeningococcus serum intravenously and intraspinally has favorably influenced the course and mortality of the disease.

XVII. ERYSIPELAS.

Definition.—An acute, infectious, endemic affection caused by the Streptococcus erysipelatis and characterized by fever, a peculiar circumscribed inflammation of the skin, and ready transmissibility.

Etiology.—Predisposing Influences.—Erysipelas is a widely prevalent disease which occurs in every climate and to which all races are liable. It is endemic at all seasons of the year and may prevail in local epidemics at any time if the conditions are favorable to its spread. Such epidemics are more common and extensive in the spring. Erysipelas belongs to the group of wound infections and spreads chiefly by accidental inoculation. Neither age nor sex therefore essentially predisposes to the disease. Incidentally certain conditions of the individual and his surroundings render him especially liable. The integuments afford less complete protection against infection at the extremes of life than at other periods. During the first two weeks, the infant is very liable to erysipelas, which most commonly starts from the umbilicus, though it may appear at any wound or abrasion. Aged persons frequently suffer from chronic diseases of the skin, such as eczema, acne, furunculosis, prurigo, varicose

ulcers and fissures where the skin and mucous membranes merge, and are hence especially liable to accidental inoculation. But these lesions may occur at any period of life. Wounds and injuries are more common in males than in females and for this reason the former suffer from erysipelas more frequently than the latter. Those who have recently undergone surgical operations and lying-in women are peculiarly liable to infection. Exhausting diseases, conditions of cachexia, chronic nephritis, and alcoholism are important predisposing factors. Among local conditions filth, overcrowding, defective ventilation, and deficient sunlight are most important. Unsanitary apartments and buildings frequently become the abiding place of the disease. The appearance of a single case of erysipelas in a surgical ward or lying-in hospital is an imperative reason not only for immediate disinfection, but also for abandoning its use for a period. Notwithstanding these precautions the disease occasionally continues to recur in modern institutions of approved construction. The greatly diminished death-rate after surgical operations and among puerperal women at the present time is largely due to the infrequency of erysipelas, and this to the scientific cleanliness of a modern technic. A family or hereditary predisposition is sometimes observed. An apparent personal predisposition is not uncommon. Certain individuals contract the disease several times, at intervals varying from some months to a year or more.

Exciting Cause.—The specific cause of the disease is the Streptococcus erysipelatis, or S. vulgaris hæmolyticus. This organism belongs to the group S. pyogenes. The Streptococcus erysipelatis is thrown off from the inflamed surface throughout the whole course of the disease and during the desquamation. It is capable of an indefinitely prolonged existence. Under ordinary circumstances it is not intensely virulent, but when a number of susceptible persons are crowded together under bad hygienic conditions the results are disastrous. It is extremely tenacious, adhering to the clothing and bedding of the patient and the furniture and walls and floor of the room occupied during his illness. It clings also to the clothing of individuals who come into contact with the patient, to the hands of operators and attendants, and to surgical instruments. By these means it may be and frequently is, in the absence of proper precautions, communicated to persons at a distance, who in their turn become centres of infection. It gains access to the organism in the vast majority of cases by demonstrable wounds or abrasions of the skin, or, less commonly, of the mucous membranes. The most minute lesion of the integument, readily overlooked or already healed when the erysipelatous flush first appears, or, if not healed, concealed by the blush itself, may serve as the point of entrance. Fissures at the angle of the mouth, nose, or eye, a lesion of the lachrymal duct, a crack in the fold behind the ear, a fissured nipple, an abrasion about the genitalia or at the anus, the prick of a needle, the piercing of the lobule for ear-rings, a scratch, in fact any solution of the continuity of the integument whatever, may be the starting point of the disease. In like manner any lesion of the mucous membrane of the upper respiratory tract or of the oropharynx may become the seat of primary infection. The condition of the mucous membrane of the genital tract in the puerperal woman especially invites infection, which is invariably

followed under these circumstances by serious results. The relationship of certain forms of puerperal infection and erysipelas is obvious. Chronic affections of the nasal or laryngeal mucous membrane, varicose ulcers and diseases of the skin render those suffering from them liable to repeated attacks of erysipelas. Abrasion of the skin for vaccination, or the use of the hypodermic syringe without proper precautions as to disinfection and cleanliness, may open the way for the erysipelatous infection.

Symptoms.—The incubation varies from a few hours to several days. Its extreme limit is not more than a week. The local and constitutional symptoms of erysipelas of the face and head and of other parts of the body are identical. When the face and head are involved it not infrequently happens that the local injury to the skin by which the infection has taken place cannot be discovered. Such cases are sometimes spoken of as idiopathic—*E. verum, E. cryptogeneticum.* When the cutaneous inflammation affects other parts of the body, a lesion of the skin or mucous membrane can almost always be found. In general terms the severity of the constitutional symptoms is in proportion to the extent and intensity of the local inflammation.

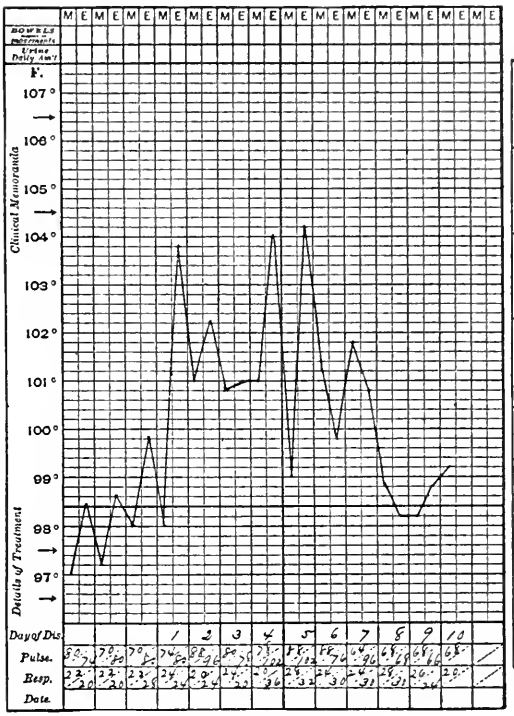


Fig. 252.—Facial erysipelas.

Prodromes are as a rule absent. The onset is commonly marked by a rigor or shivering, followed by a rapid rise of temperature to 103°–105° F. (39.5°–40.5° C.). Headache, pains in the back and limbs, nausea and vomiting also frequently occur. In the course of some hours the signs of local inflammation appear. In many of the milder cases the constitutional symptoms are at first slight and the patient becomes aware of the trouble by burning and pricking sensations in the affected skin. The skin is reddened, tense, and glossy. It is swollen and œdematous and the borders of the affected area are abruptly marginate. It is hot and firm to the touch and the patient complains of subjective sensations of burning, tension, and stiffness. It is a peculiarity of the erysipelatous inflammation that, involving a limited area at first, it tends to spread broadly in various directions, advancing by a well-defined border slightly raised above the level of the surrounding skin. This advance is usually from an ear across the

face to the other ear, or from any point, as the corner of the nose or mouth, or the canthus of an eye, rapidly over the entire face, into the hairy scalp, and downwards to the neck. The general swelling in severe cases is marked and the loose skin of the eyelids and adjacent parts becomes enormously œdematous. The eyelids cannot be opened, the nose is swollen to an extraordinary bulk, the lips hugely distended, the ears cushiony and deformed and the whole countenance strangely disfigured and unrecognizable. As the inflammation advances cord-like thickenings of the lymphatic vessels may often be felt upon palpation beyond its border in the area of skin which as yet presents no discoloration or œdema. In some instances the involved lymphatic vessels appear as reddened strands or spots, advanced areas of infection which are speedily overtaken by the progressing inflammation. The neighboring superficial lymphatic glands are very often enlarged and tender. In severe cases vesicles form upon the surface of the inflamed skin, especially upon the eyelids, ears, and forehead. In the course of three or four days the inflammation reaches its height and begins to undergo resolution at the point first involved. Here the color becomes paler, the swelling diminishes, and desquamation takes place; meanwhile the peripheral inflammation may for a day or two continue to advance. Careful inspection from day to day reveals the fact that at any given point the inflammation reaches its maximum in the course of three or four days and then rapidly subsides, a matter of importance in estimating the worth of local therapeutical applications. The mucous membrane of the mouth and nose is frequently involved by extension. The mouth and gums are reddened, the pharynx is congested, the tongue swollen, dry, and cracked. The pulse is rapid. The mind is commonly clear. In the course of six or seven days the rash in favorable cases ceases to spread, the redness and swelling subside, the temperature falls by crisis, and the patient enters upon convalescence. There is marked leucocytosis. The urine is scanty and high colored. Febrile albuminuria is commonly present. Recrudescences of fever frequently occur. Relapses are not common.

Anomalies in the clinical course relate to the rash and to the constitutional disturbances. That form in which vesiculation is abundant is known as *E. vesiculosum;* that in which bullæ form as *E. bullosum.* The contents of the vesicles and blebs is usually a slightly turbid serum. Pus may be present—*E. pustulosum.* These lesions may be ruptured by accidental violence or the contents may undergo gradual resorption. Thin, yellowish-brown crusts result, which after a little time separate without scar formation. Deeper abscess formation in the connective tissue is not uncommon—*E. phlegmonosum.* A very grave form is that in which, in consequence of enfeebled powers of resistance, the swelling and tension result in necrosis and gangrene of the skin—*E. gangrænosum.* The inflammation in rare cases shows a remarkable tendency to spread—*E. migrans.* The inflammation may advance from the face over the neck and chest, subsiding in one area as it extends to another, until it has traversed the greater part of the body. The duration of the disease may extend over many weeks, and death may occur from exhaustion or from a complicating pneumonia. Anomalies in the constitutional symptoms consist in the absence

of fever—*E. afebrile* in hyperpyrexia which is apt to terminate fatally with progressive cardiac weakness and coma; or in great constitutional depression from the outset, a condition to which cachectic and aged persons and those given to excesses in alcohol are especially liable. Erysipelas of the newborn, starting at the navel, shows an abruptly marginate area of redness and induration, which may be superficial but commonly involves the deeper tissues. It extends rapidly and may invade the greater part of the trunk. Suppuration and gangrene sometimes occur. The prognosis is ominous.

Complications and Sequels.—The visceral complications are due to general septic infection. Purulent meningitis may occur in erysipelas of

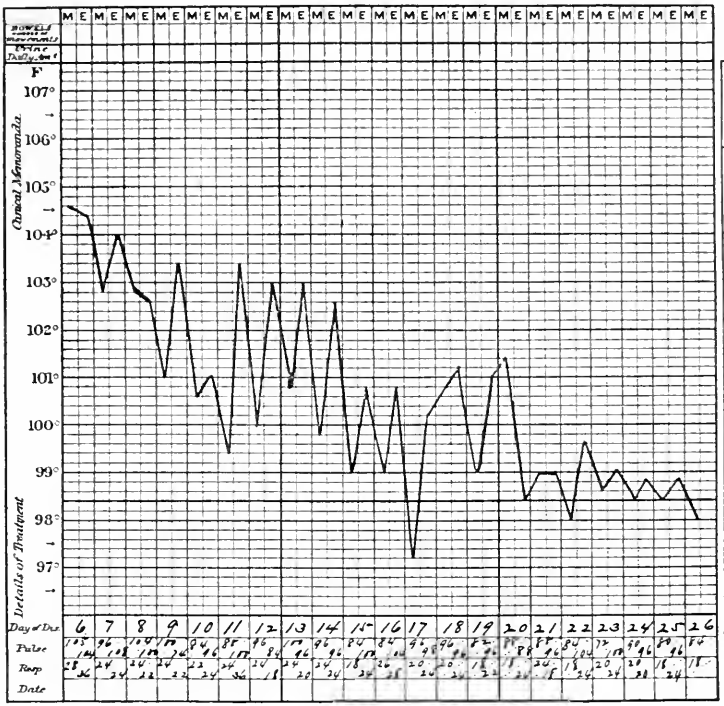


Fig. 253.—Erysipelas ambulans. Woman aged 50. Recovery.

the face and head. When the inflammation involves the mucous membrane of the throat or invades the neck, acute œdematous laryngitis may occur and prove rapidly fatal. Croupous pneumonia is sometimes encountered. Bronchopneumonia is a common late complication in fatal cases. Otitis media and acute nephritis are occasional complications. Septic inflammation of one or more joints may occur. Malignant endocarditis, pericarditis, and pleurisy are important complications. Among the sequels are areas of cutaneous hyperæsthesia or anæsthesia and persistent neuralgias. Repeated attacks of erysipelas have in rare instances been followed by thickening and induration of the skin. The hair falls out after erysipelas of the scalp, sometimes after severe attacks involving distant parts of the body, as in the other acute infections.

Diagnosis.—The DIRECT DIAGNOSIS of erysipelas rarely presents difficulty. It rests upon the sudden onset, the chill or shivering, fever, and other constitutional symptoms, and the peculiar character of the inflammation of the skin, in which rapid advance from an infected centre, elevation above the level of the surrounding skin, distinct margination, and the tendency to resolution in the region first involved while evolution is taking place at the border, are characteristic.

The DIFFERENTIAL DIAGNOSIS between erysipelas and other forms of dermatitis can be easily made. The main fact is that erysipelas is an acute infectious process attended with fever which begins abruptly and ends by crisis.

Prognosis.—Previously sound individuals except at the extremes of life usually recover. In the new-born erysipelas is commonly fatal, in the aged very often so. In broken down and cachectic persons and drunkards the prognosis is unfavorable. Death is usually the result of intense general infection or grave complications. Erysipelas is not only less frequent, but it is also much less fatal than formerly. The mortality is about five per cent. It is higher in hospitals than in private practice.

XVIII. SEPSIS.

Septicæmia; Pyæmia; Septicopyæmia.

Definition.—A disease produced by the general invasion and growth in the body of pyogenic micro-organisms.

Pyæmia is the term used to designate the condition in which pus collections occur in various parts of the body in consequence of the lodgement of infected emboli; *septicæmia*—*bacteræmia*—that condition in which purulent collections are absent, but with or in the absence of a local infection there is invasion of the blood and tissues by bacteria together with the signs of profound disorder of the entire organism, and *septicopyæmia*, the condition in which bacteræmia and pyæmia are present at the same time. *Sapræmia* is the condition caused by the absorption into the blood of septic or putrid products. The general term *septic infection* or *sepsis* is more convenient and more in accordance with the facts. Some other definitions are necessary. *Infection* is the term used to designate the pathological processes caused by the implantation and growth of pathogenic micro-organisms, most of which are specific. *Toxæmia* is used to denote, (a) the presence of soluble toxic substances or toxins in the blood, mostly elaborated by pathogenic micro-organisms in their growth and multiplication, and (b) the morbid processes which those poisons produce.

Septicæmia and toxæmia are sometimes associated, as in enteric fever, in which the pathogenic organism—Bacillus typhosus—may be grown in cultures from the blood, while the symptoms of the disease are those of a continuous intoxication; sometimes separate, as in tetanus and diphtheria, in which the infection is local and the pathogenic organisms develop in circumscribed areas, while their poisonous products produce characteristic constitutional effects.

Etiology.—Predisposing Influences.—The predisposition to septic infection is general. It occurs everywhere, at all periods of life, and under the most varying circumstances. Pathogenic Agents.—The pyogenic cocci—streptococci and staphylococci—are the most common. Other organisms capable of causing sepsis are Micrococcus lanceolatus (pneumococcus), gonococcus, Bacillus coli, Bacillus typhosus, Bacillus proteus, Bacillus pyocyaneus, and Bacillus influenzæ.

Symptomatology.—The symptom-complex is that of a severe constitutional disease. In many of the cases the general symptoms are simply those of any intense infective process, without, however, definite signs of visceral or other localization. In another large group of cases to the foregoing symptoms are added those of an inflammatory implication of the endocardium or pericardium with which the evidences of myocardial changes are shortly associated. Again, the evidences of inflammation of the bones and joints dominate the situation. In a very extensive group of cases

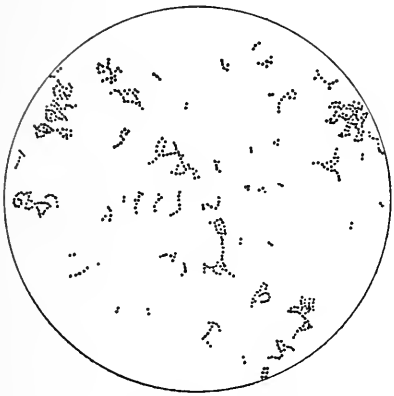


Fig. 254.—Streptococcus pyogenes.

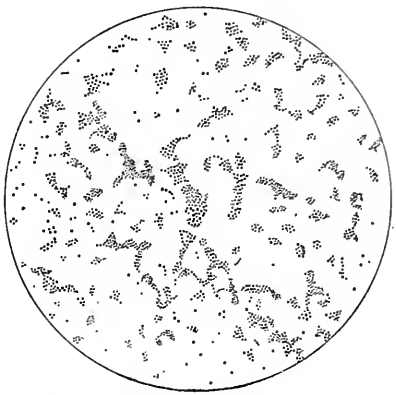


Fig. 255.—Staphylococcus pyogenes aureus.

the tegumentary structures are especially involved in septic inflammatory processes, such as forms of erythema, scarlatiniform eruptions, malignant erysipelas, and acute septic phlegmon. Subcutaneous hemorrhages are common. Finally, we recognize a great group of cases in which inflammatory and suppurative disease of the various viscera, as for example, the lungs, kidney, liver, stomach, and intestines or spleen, give rise to the chief manifestations of the septic process.

The onset may be gradual, with chilliness or chills, general malaise, dragging pains in the limbs, and irregular fever. More commonly a decided rigor initiates an active febrile movement. Pallor, faint cyanosis, rapid and feeble pulse, anorexia, constipation alternating with diarrhœa, a tendency to profuse sweating, and a mind strikingly clear and alert are symptoms encountered in well-developed sepsis. Soreness of the bones and muscles, tender joints, great irregularity of temperature, with wide oscillations and a profound feeling of lassitude are also common. A leucocytosis of varying degree, sometimes high, occurs in the majority of cases. In the graver cases even with abscess formation at various points, an increase of leucocytes may be absent.

Toxæmia—Local Infection with the Absorption of Toxins.—Familiar examples of mild and transient forms are the chilliness and fever which attend an attack of angina tonsillaris or a local phlegmonous inflammation. Most important are erysipelas, diphtheria, and tetanus, diseases in which the pathogenic organisms develop locally, while the constitutional symptoms are caused by the absorption of the poisonous products of their growth.

Septicæmia; Bacteræmia—Local Infection with the Invasion of Micro-organisms.—Streptococcus and staphylococcus infection is commonly at first local, the toxins undergoing absorption and causing symptoms—toxæmia. The process may be arrested at this point; if not, the cocci enter the blood stream and are carried to all points of the body without causing foci of suppuration. The cause of the infection may frequently be traced, as in puerperal sepsis or injuries of the extremities, along the lymphatic vessels.

The case now becomes more severe; the symptoms more urgent. Specific infections in which a primary local infection may become generalized are gonorrhœa, pneumonia, and puerperal fever. To a slight extent the micro-organisms of diphtheria may also enter the blood stream. In the severer forms of the specific infectious diseases, as scarlet fever, diphtheria, enteric fever, and tuberculosis, secondary or mixed infections frequently take place, greatly adding to the gravity of the case and often obscuring the features of the primary disease. The most active agent is the streptococcus.

Cryptogenetic Septicæmia—General Septicæmia in the Absence of Local Infection.—Cryptogenetic septicæmia may develop in the subjects of acute or chronic disease or even in persons in apparently good health. This condition explains a considerable number of obscure febrile cases. Many of the cases are terminal infections. No focus of infection is apparent during life or demonstrable after death. The streptococcus is the common infecting organism, but the staphylococcus, pneumococcus, B. proteus and B. pyocyaneus may be found.

Septicopyæmia; Pyæmia—General Infection with Suppurative Foci.—The organisms may colonize in various parts of the body and give rise to abscess formation. These suppurative processes are due to the lodgement of infected emboli. The lesions are known as embolic or metastatic abscesses. In infected wounds, septic phlegmon, and osteomyelitis they are frequently encountered in the lungs; in suppurative lesions in the intestines, or elsewhere in the parts tributary to the portal system, metastatic abscesses occur in the liver and may be accompanied with suppurative pylephlebitis. Endocarditis is of common occurrence in septicæmia. The most common organisms in septicopyæmia are streptococci and staphylococci.

Terminal Infections.—Secondary or terminal infection is the cause of death in many acute and chronic diseases. The infection may be local and take the form of visceral disease. The serous membranes are especially liable to these terminal inflammations, and acute pleurisy, pericarditis, peritonitis, endocarditis, or meningitis are final events in many cases of nephritis, arteriosclerosis, cirrhosis of the liver, and cerebral and spinal

disease. Not rarely the terminal process is an acute miliary tuberculosis. The enterocolitis so common toward the end of chronic diseases may be classed with the terminal infections. Less frequently the terminal infection is general. This especially occurs in chronic renal and cardiac disease, tuberculosis, leukæmia, and Hodgkin's disease.

Diagnosis.—Direct Diagnosis.—It cannot be made in the beginning of the milder cases. When the symptoms are severe or the illness has lasted for some time, the history of an abortion or confinement, an infected wound involving the skin or mucous membrane, an attack of tonsillitis and especially middle-ear disease, and the occurrence of chills, irregular fever, copious sweating, progressive pallor, and asthenia render the diagnosis probable. Gonorrhœal infection, the reinfection of old scars, and the previous occurrence of disease of the bones, periosteum, or joints are also of diagnostic significance. The signs of an endocarditis are important, especially if the murmurs undergo changes in the course of a short time or are associated with embolic abscesses. Blood cultures may not show the presence of pathogenic micro-organisms.

Cryptogenetic sepsis is more difficult of diagnosis. The symptom-complex is not characteristic. Chills and irregular fever of wide range, with irregular, even prolonged intermissions, copious sweating, profound asthenia, pallor, emaciation, followed after intervals by visceral inflammations, especially affecting the heart, rapid respiration, circumscribed patches of pulmonary consolidation, pleural effusions, enlargement of the liver, localized bone disease or arthritis, and polymorphous cutaneous lesions, especially erythema and hemorrhage, are suggestive. The diagnosis must in many instances be made by exclusion.

Differential Diagnosis.—*Acute Miliary Tuberculosis.*—The general bronchopneumonia affecting the whole of one or both lungs which is characteristic of this disease is not seen in sepsis. If circumscribed tuberculous foci are present, the diagnosis of miliary tuberculosis is probable. Implication of the serous membranes, the pleuræ, pericardium, meninges, or peritoneum increases the resemblance to sepsis and renders the diagnosis more difficult. *Chronic Ulcerative Phthisis.*—In the gravest cases and particularly in the *stadium ultimum*, symptoms occur which are not caused by the Bacillus tuberculosis. These secondary infections are due to streptococci and are mostly terminal. Other organisms are present in the sputum. *Influenza.*—Severe cases of influenza may give rise to difficulties in diagnosis, especially in those cases in which pneumonia, bronchopneumonia, pleurisy, and local pus formation occur. The epidemic prevalence of influenza, the sudden onset with characteristic symptoms, the prominence of catarrhal symptoms in the majority of instances, and the severe neuralgias early in the attack are of diagnostic value. *Enteric Fever* (see p. 30). *Malaria.*—Perhaps no more common error in diagnosis occurs. In phthisis, in internal abscess, in suppurative disease of the liver or hepatic fever from impacted calculus, in malignant endocarditis, the recurrent chills, irregular high temperature, and profuse sweating too often betray the incautious practitioner into a false diagnosis of malaria. The presence of the malarial parasite in the blood and the therapeutic test, or either of them, are conclusive.

Prognosis.—The prognosis is ominous. Very mild cases recover. Many cases make an apparent recovery, only to recur. The fulminant cases are fatal in a short time. Of visceral localizations, septic endocarditis is the most grave. In the absence of local abscess formation recovery may, in rare instances, take place after very grave constitutional symptoms have lasted a long time. Brilliant results sometimes follow the early evacuation of pus and effectual drainage. All forms of focal infection are to be considered.

XIX. RHEUMATIC FEVER.

Acute Rheumatism; Acute Polyarthritis.

Definition.—An acute febrile disease due to streptococcus infection, characterized by acid sweats, multiple polyarthritis of fugacious character and a tendency to endocarditis and less frequently pericarditis. It usually follows acute or subacute angina and is not contagious.

Etiology.—Predisposing Influences.—Rheumatic fever is a disease of northern and temperate climates. Cold and dampness, and especially a combination of these two seasonal conditions, constitute predisposing factors of great importance. Rheumatic fever is rare in the first years of life and after the age of fifty. Adolescents and young adults are especially liable. The two hemidecades of greatest liability are those from 15 to 20 and from 20 to 25. First attacks are very rare after the fortieth year. The liability of the sexes is much modified by age and occupation. If these factors are disregarded males appear to be more liable than females in about the proportion of 2.5 to 1.0. Under twenty the disease is more common in females. The predisposition appears to be hereditary, usually from the maternal side. A great majority of the cases occur among coachmen, cooks, bakers, housemaids, sailors, gardeners and outdoor laborers.

Exciting Cause.—The causal relation of the diplococcus described by Poynton and Paine (1900), to which they gave the name *Diplococcus rheumaticus* or *Streptococcus rheumaticus,* was until recently generally admitted. The later studies of Rosenow are of great importance. This investigator isolated from joints, tonsils and lymph-nodes, streptococci, which by special methods of culture and transmission by serial inoculations into animals, he claims may be developed by gradation into typical hemolytic streptococci on the one hand and into pneumococci on the other, with characteristic pathogenic selective affinities. The inoculation of these organisms into animals has been followed by polyarthritis, myocarditis and myositis. The phenomena produced by the inoculation of various indifferent streptococci present only a superficial resemblance to rheumatic fever. The hypothesis that the infection is septic rather than specific was thought to find support in the character of the fever, the joint affection, the tendency to implication of serous membranes, the sweating, anæmia, leucocytosis, and the tendency to relapse. On the other hand, pyæmic joints undergo suppuration and pursue a wholly different course, and the pains of sepsis are not influenced by the salicylates. The chemical, metabolic, and nervous hypotheses have merely a historical interest.

Symptoms.—Prodromes are not common. When present they consist of sore throat, slight pains in the joints, and malaise. Not rarely a well-marked attack of angina tonsillaris precedes the joint affection.

The onset is usually abrupt. There is very often the chilliness which attends the development of a mild infective process. Fever of moderate intensity—101°–103° F. (38°–39.5° C.)—and irregular type follows and in the course of twenty-four or thirty-six hours the nature of the attack is established. One or more, usually several, joints are now swollen, reddened, and painful. The pulse is frequent, full, and soft. The tongue is covered with a soft, thick, white coating; appetite is lost; thirst, constipation, and scanty, high-colored, and very acid urine occur. There is frequently abundant, highly-acid, and ill-smelling perspiration. The joints are involved successively but without regular order. The large joints, as the knee, ankle, shoulder, are most frequently affected; the smaller joints of the hands and feet somewhat less so. The wrists and ankles are often enlarged and exquisitely tender and painful from the simultaneous implication of many joints and the sheaths of the tendons. The arthritis is curiously fugacious. As one joint is attacked the inflammation subsides in another previously involved. This constitutes a characteristic clinical feature of the disease. The inflammatory exudate is endo- and periarticular. Suppuration does not occur and ankylosis is very rare, being not a phenomenon of rheumatic fever but a secondary process from want of use and fixation. In severe and protracted cases numbers of joints are implicated and the vertebral articulations do not always escape.

Pain is a constant and conspicuous symptom. It is spontaneous and usually agonizing upon movement and pressure. Frequently the weight of the sheet cannot be borne. Prostration, inability to sleep, and abject helplessness add to the sufferings of the patient. The temperature range does not often exceed 103° F. (39° C.) and rarely, except in hyperpyrexia, surpasses 104° F. (40° C.). It does not conform to type and is extremely irregular, with marked remissions and exacerbations. The defervescence is by gradual lysis. Recrudescences are common and relapse frequently occurs. Anæmia of high grade develops with great rapidity. A leucocytosis of moderate degree is present. Febrile albuminuria is common. The saliva is sometimes acid in reaction. There are subacute forms with less intense symptoms which sometimes tend to become chronic. In children rheumatic fever may be attended with very slight or obscure joint affection but with marked and disabling heart lesions. The course of the disease is very variable, the duration being commonly from four to six weeks, with remissions and exacerbations. Subacute and transient attacks recur. The attack does not confer immunity against subsequent attacks: on the contrary, like croupous pneumonia, diphtheria, and erysipelas, rheumatic fever tends to recur and many persons in the course of time experience several attacks.

Rheumatic Hyperpyrexia; Cerebral Rheumatism.—In rare cases a day or so after the onset, but usually during the course of the second week, a rapid rise of temperature to 108°–110° F. (42.5°–43.5° C.) occurs. Delirium, stupor, a feeble, frequent, and flickering pulse, and extreme prostration accompany the hyperpyrexia, In the course of a few hours the

patient usually falls into a comatose state. This form of rheumatic fever is almost always fatal. If the temperature is reduced by cold baths or external cold it rises again. In some instances convulsions precede the coma. Rheumatic hyperpyrexia is extremely rare in this country.

Heart.—Endocarditis, pericarditis, and associated myocardial changes are so frequent that they must be regarded as pathological processes incident to the disease rather than accidental complications. The incidence is variously estimated at 33 to 50 per cent. It is probably higher than these figures indicate. *Endocarditis.*—This is by far the most common of the heart affections and rheumatic fever is by far the most common cause of chronic valvular disease. The liability is greatest in the rheumatic fever of childhood and decreases with age. On the other hand it increases with the number of attacks. The mitral valve system is most frequently involved, the aortic next, and both next, the ratio being about 90–25–20. Ulcerative endocarditis is of infrequent occurrence in rheumatic fever. *Pericarditis.*—Pericardial inflammation may be associated with endocarditis, as is commonly the case, or occur independently. Well-marked pericardial signs may mask an indistinct endocardial murmur, which very often becomes plainly audible as the friction sounds subside. The exudate may be fibrinous, serofibrinous, or purulent. The last occurs more frequently in childhood. *Myocarditis.*—Changes in the heart muscles—granular and fatty degeneration—are associated in varying degree with the endocarditis and pericarditis, and manifest themselves clinically by enfeebled action and the signs of dilatation.

Lungs and Pleuræ.—Pleurisy may occur and the exudate is often serofibrinous, the effusion not, however, commonly attaining a great volume. Pneumonia is an occasional complication. Acute pulmonary congestion is a grave accident.

Nervous System.—Delirium, stupor, convulsions, and coma arise in the cases of hyperpyrexia and are sometimes manifestations of uræmia. Delirium may be due to the salicylates or other drugs. The mental condition in rheumatic fever is, as a rule, even in severe cases, remarkably clear. Chorea, while it does not often appear during the attack of rheumatic fever, follows it in about 15 per cent. of the cases.

Cutaneous Affections.—These occasionally appear, as in the other acute febrile infections. They are not important and comprise sudamina, miliaria, urticaria, forms of erythema and petechiæ. *Subcutaneous nodules* occasionally develop upon the tendons and fasciæ, about the wrists and hands, and elsewhere. They vary in size up to that of a pea. They grow rapidly and slowly disappear. They are not usually tender to the touch, nor painful.

Diagnosis.—Direct.—The direct diagnosis of rheumatic fever is not usually attended with difficulty. It rests upon the association of the foregoing symptoms, especially the rapid onset, the fugacious polyarthritis, irregular fever, abundant acid sweats, rapidly developing anæmia, and tendency to cardiac complications.

Differential.—1. *Sepsis; Septicopyæmia.*—Arthritis, irregular fever, and endocarditis are common to both diseases. But in septic conditions

the arthritis is fixed, not fugacious, affects a few joints, not many, and tends to suppuration and disorganization instead of *restitutio ad integrum* as in rheumatic fever. The fever in sepsis is as a rule more distinctly intermittent, with higher maxima, and is interrupted by periodical chills sometimes of ague-like regularity. The endocarditis of sepsis is severe, often malignant, with embolic phenomena and retinal hemorrhages. Cases occur in which for a time the differential diagnosis between relatively mild septicopyæmia and severe rheumatic fever cannot be positively made. 2. *Acute Osteomyelitis.*—When the lower end of the femur or the tibia is affected the differential diagnosis may be at first obscure. In the rare cases in which several bones are involved the resemblance to rheumatism is increased. The epiphysis is the seat of the disease rather than the joint, and the local and constitutional symptoms are more severe. 3. *Acute Arthritis of Early Infancy.*—The knee or the hip is usually affected. The affection is mostly monarticular and goes on to early suppuration. It is commonly pyæmic; sometimes gonorrhœal. 4. *Gonorrhœal Arthritis* (see p. 230). 5. *Gout.*—Many cases of podagra are falsely diagnosticated as rheumatism. An arthritis confined to one or two joints, especially the metatarsophalangeal joint of the great toe, the knee, or the ankle, of extremely acute onset and great intensity, with cyanotic redness of the skin which is tense and glossy, exquisitely painful both at rest and on movement, and so tender that the weight of the bedclothes can scarcely be borne, speaks for gout, especially if tophaceous masses are present in the helix of the ear or around the small joints and the patient has reached middle age. 6. *Arthritis Deformans.*—The acute outbreaks of joint inflammation by which certain forms of this disease advance cannot be differentiated from rheumatic fever in the early course. There is fever, together with redness, swelling, tenderness and pain, mostly affecting the small joints. When these symptoms pass, however, there remains the evidence of changes in the joints and periarticular thickening. Fresh attacks of more or less intense arthritis occur and after each one the signs of damage to the joints are more pronounced. 7. *Meningitis.*—When in rheumatic fever the vertebral articulations are involved, there may be severe pain upon movement of the neck, together with painful rigidity of the muscles. As fever is present, the condition, especially in the absence of joint affection of the extremities, may closely simulate meningitis. The absence of severe headache, pupillary derangements, hyperæsthesia, Kernig's sign, and negative results upon examination of the fluid obtained by spinal puncture are of diagnostic importance. 8. *Peliosis Rheumatica.*—The multiple arthritis and fever of Schönlein's disease may suggest rheumatic fever. The simultaneous appearance of purpura, purpura urticans, and erythema exudativum, especially when associated with hemorrhage from mucous surfaces or the evidence of internal bleeding, is decisive. 9. *Hysteria.*—A hysterical arthritic neurosis, usually involving the knee, elbow, or wrist, does not often closely simulate rheumatic fever.

Prognosis.—The course of rheumatism varies from two or three to six weeks or longer and is marked by many remissions and exacerbations, both of the fever and other constitutional symptoms and the arthropathy.

Rheumatic fever tends to recovery. The mortality does not exceed 2 or 3 per cent. and death is the result not of the disease in its ordinary manifestations but of the heart affection or hyperpyrexia. It acquires, however, a sinister importance on account of the frequency of the implication of the heart, as the result of which arise initial lesions of the valves and myocardium, especially progressive, constituting the conditions of deformity and impaired function known as chronic valvular disease, irreparably damaging to function and ultimately the cause of death.

XX. YELLOW FEVER.

Definition. — A febrile disease of tropical and subtropical countries due to the filtrable virus of Reed, Carroll and Agramote, transmitted by the bite of a variety of mosquito—*Stegomyia calopus fasciata*—and characterized by jaundice, albuminuria, slow pulse, and black vomit.

Etiology. — Predisposing Influences.—Yellow fever has frequently been transported to the seaboard cities of the United States and toward the end of the eighteenth century prevailed in frightfully disastrous epidemics in Philadelphia and other northern cities. It is a disease of the seaboard and low levels. It rarely shows itself above an altitude of 1000 feet. It occurs chiefly in cities and, during outbreaks, is most prevalent in the low, badly drained, and overcrowded districts occupied by the poor, and in the hot season. The epidemics in the United States have always appeared during the summer and autumn and come to an end upon the occurrence of frost.

The Actual Cause.—The following are the conclusions of the Yellow Fever Commission of the United States Army:

1. The mosquito—*Stegomyia fasciata* (Fig. 256)—serves as the intermediate host for the parasite of yellow fever. 2. Yellow fever is transmitted to the non-immune individual by means of the bite of the mosquito that has previously fed on the blood of those sick with this disease. 3. An interval of about twelve days or more after contamination appears to be necessary before the mosquito is capable of conveying the infection. 4. The bite of the mosquito at an earlier period after contamination does not appear to confer any immunity against a subsequent attack. 5. Yellow fever can also be experimentally produced by the subcutaneous injection of blood taken from the general circulation during the first and second days of this disease. 6. An attack of yellow fever produced by the bite of the mosquito, confers immunity against a subsequent attack of the non-experimental form of this disease. 7. The period of incubation in thirteen cases of experimental yellow fever has varied from forty-one hours to five days and seventeen hours. 8. Yellow fever is not conveyed by fomites, and hence disinfection of clothing, bedding, or merchandise, supposedly contaminated by contact with those sick with this disease, is unnecessary. 9. A house may be said to be infected with yellow fever only when there are present within its walls contaminated mosquitoes capable of conveying the parasite of this disease. 10. The spread of yellow fever can be most effectually controlled by measures directed to the destruction of mosquitoes

and the protection of the sick against the bites of these insects. 11. While the mode of propagation of yellow fever has now been definitely determined, the specific virus has not yet been cultivated.

Symptoms.—The period of incubation is three or four days, sometimes longer. In 13 experimental cases it varied from 41 hours to 5 days and 17 hours. The course of the attack may be divided into a stage of invasion, a stage of remission and a stage of collapse. These periods are, however, not always well characterized. 1. Invasion.—The onset is sudden, without prodromes, and commonly in the early morning. It is marked by chilliness, headache, severe pains in the back and limbs, a rapid rise of temperature to 102°–105° F., and pungent heat and dryness of the surface. The tongue is moist and covered wth a thick white fur. There is usually some soreness of the throat, together with nausea and vomiting, which become more severe upon the second and third days, and constipation.

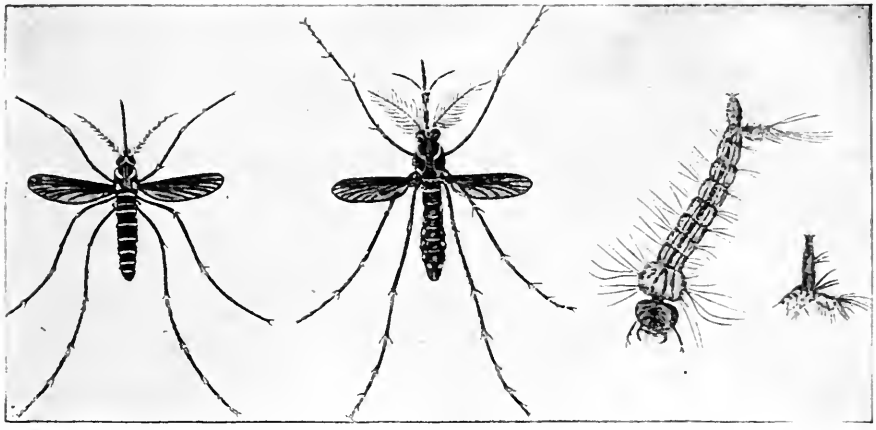


Fig. 256.—*a*, *Stegomyia fasciata*. Fabr. Male. (Magnified.) *b*, *Stegomyia fasciata*. Fabr. Female. (Magnified.) *c*, Mature larva of *Stegomyia fasciata* on the left, respiratory siphon of *Culex* to the right (magnified).

The facies even upon the first day is suggestive, even characteristic. It is flushed and there is slight tumefaction of the eyelids and lips. The conjunctivæ are injected and icteroid. Later the intense jaundice from which the disease takes its name rapidly invades the entire surface. The fever having attained its fastigium during the first day maintains its elevation for two or three days and subsides in favorable cases by lysis. In abortive cases the temperature may fall to normal in twenty-four or thirty-six hours. 2. The Remission or Stage of Calm.—This period lasts two or three days. The symptoms ameliorate and the condition of the patient is in every way more satisfactory. Convalescence may now set in with rapid improvement, or there may be febrile reaction lasting from one to three days and terminating in rapid lysis, or the patient may pass into: 3. The Stage of Collapse.—This period is attended with characteristic "black vomit" and other hemorrhages. The vomiting, in the grave cases, is uncontrollable and copious, being attended with great abdominal pain and exhaustion. The oozing of blood from the mucous surfaces and the occurrence of petechiæ usually precede death.

The pulse upon the first day does not usually exceed 110 per minute and, notwithstanding the persistence of a relatively high temperature, becomes during the second or third day progressively slower until it may reach, with a temperature of 102°–103° F., a rate as low as 50, 40, or even 30 per minute. This low pulse-rate, with a persistent or even rising febrile movement, is a characteristic and striking feature of the disease. Albuminuria occurs about the third day of the attack. In the mild cases it is transient, but in the severe cases it is continuous, abundant, and accompanied by the ordinary signs of acute nephritis. Suppression may occur and the manifestations of uræmia, convulsions and coma, or these in alternation, lead to a rapidly fatal issue. Delirium may be present early in the course of severe cases. The mental condition is usually, however, one of remarkable clearness and alertness.

Varieties.—1. *Mild cases*—"walking yellow fever"—present simply a transient fever and slight jaundice and would not be recognized except in the light of the prevalent epidemic. These cases are especially dangerous, since they may be the source of contamination of mosquitoes and the subsequent infection of non-immune persons with the fever in its severer forms. 2. *Average cases with high fever* and the characteristic features of the infection—jaundice, vomiting, fever, slow pulse, albuminuria, black vomit and other hemorrhages, and prostration. 3. *Malignant.*—The patient is overwhelmed by the infection and death occurs in the course of the second or third day.

Convalescence in favorable cases is rapid and complete, the albuminuria usually passing away in the course of a little time. In severe cases terminating in recovery, the convalescence may be protracted by parotid bubo, suppurative processes elsewhere, or persistent diarrhœa. Second attacks are exceedingly rare.

The screening of all cases of yellow fever and the general use of screens for protection against malaria, systematic drainage and oil on all standing water about both dwellings and working localities have eliminated both *Stegomyia* and *Anopheles* and brought to an end the prevalence of yellow fever and malaria in Havana, Porto Rico and the Canal Zone.

Diagnosis.—Direct Diagnosis. — The symptom-complex in well-developed cases is so characteristic that a positive diagnosis would appear to be a simple matter, especially when a number of cases have occurred in a circumscribed region. Commercial interests and considerations of local policy, have, however, in many instances, interposed insuperable difficulties to the recognition of the early cases—difficulties that have frequently led to wide-spread and disastrous epidemics. In some such instances the disease has been reported as dengue, in others as malarial fever.

Differential Diagnosis.—*Dengue.*—The facies, jaundice, albuminuria, slow pulse, great severity, and high mortality clearly differentiate yellow fever from dengue. The difficulties relate to the initial cases, which may be mild, and the fact that the two diseases may coexist in the same locality. Every suspect should be at once isolated in a screened hospital. *Malaria.*—The differential diagnosis concerns the estivo-autumnal variety which especially prevails in the regions and at the season of the year in

which outbreaks of yellow fever are liable to occur. The facies, early jaundice, early albuminuria, slight enlargement of the spleen, hemorrhages, especially black vomit, late bradycardia, and the absence of the blood parasite justify the diagnosis of yellow fever. In estivo-autumnal fever the facies is not characteristic, jaundice and albuminuria are later, the splenic tumor is more marked, black vomit and bleeding gums are wholly exceptional. In hemorrhagic malarial fever, hæmaturia, a rare symptom in yellow fever, is most conspicuous.

Prognosis.—The mortality ranges from 10 to 80 per cent. It varies greatly in different epidemics. Among the working classes and hard drinkers it is especially high. Of favorable prognostic significance are mild fever, slight jaundice, a free secretion of urine, and the absence of black vomit. High fever at the onset is ominous. Black vomit, though serious, is not invariably followed by death. Suppression of urine and uræmic symptoms are rarely followed by recovery.

XXI. CHOLERA.

Cholera Asiatica; Cholera Infectiosa.

Definition.—An infectious disease, endemic and epidemic in certain districts of India, and occasionally epidemic in Europe and America, caused by the comma bacillus of Koch—*B. cholera asiaticæ*—and characterized by violent purging, rice-water discharges, and early collapse.

Etiology.—Predisposing Influences.—Of great importance is exposure in an infected district, but the chief danger lies in the drinking of water contaminated with the fecal discharges of cholera patients. Cholera is endemic upon the delta of the Ganges. Thence it is from time to time transported along the lines of commerce to various parts of the world. Cases on ship board have reached the New York Quarantine Station on several occasions in the last half-century, but the disease has not gained foothold upon our shores since 1873. It has prevailed extensively in the East in recent years and is still to some extent epidemic in the Philippines. Outbreaks are more common in warm climates—India, Egypt, the Islands of the Malay Archipelago—but the disease has prevailed fiercely in Siberia and Northern Russia and to some extent in Canada. Warm weather favors the spread of cholera, but cold does not arrest it. It is especially a disease of seaport cities and commercial centres, being transported by persons and effects. It journeys in the East with caravans and pilgrims. It is not conveyed by the atmosphere and does not advance at a faster rate than that of ordinary commercial intercourse. In epidemics those who handle the soiled linen of the sick or remove the discharges are especially liable to contract the disease. Physicians and nurses on the contrary are seldom attacked. Students in the study of the germs have contracted "laboratory cholera." The drinking of contaminated water or milk, articles of uncooked food as salads and the like washed with such water, other articles of food accidentally contaminated, are common causes of the disease. The part played by the house-fly in mechanically transporting the pathogenic organism from the stools to articles of food is most important. Every period of life is liable.

Exciting Cause.—The "comma bacillus," discovered by Koch in 1884, is the cause of the disease. This organism is present in all cases of Asiatic cholera and does not occur in other diseases. It is a spirochæta, morphologically appearing as a slightly curved rod, about half the length of the tubercle bacillus but much thicker than that organism, sometimes presenting an S-shaped appearance, and occasionally assuming spiral curves. As other organisms present similar forms, the characteristic growth in cultures becomes important. Comma bacilli are found in the stools from the onset of symptoms and in the rice-water discharges and contents of the intestine after death in almost pure culture. They are rarely present in the vomited material and then only after violent or protracted retching. They are not present in the circulating blood or in the viscera, but are sometimes found in the intestinal glands and submucosa. They have been demonstrated in water tanks and in other drinking water supplies during epidemics. The symptoms are due to a virulent toxin, caused by the bacilli, which acts chiefly upon the vasomotor system. The immunity of certain persons during epidemics, and the fact that virulent cholera bacilli have been isolated from the stools of healthy individuals raises the question as to natural immunity. Artificial immunity can be established in the cases of laboratory animals and human immunity by the methods of Haffkin. General epidemics in a community are caused by contamination of the water supply and usually arise with great rapidity. Circumscribed outbreaks develop more slowly and the source of the infection cannot always be traced—contact infection, bacillus carriers.

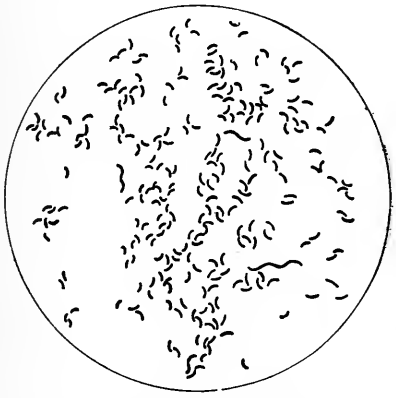


Fig. 257.—Bacillus of Asiatic cholera.

Symptoms.—The period of incubation varies from two to five days. The course of the attack may be divided into four stages. Any one of these stages may be absent.

1. Premonitory Diarrhœa.—Looseness of the bowels may begin abruptly or be preceded by colicky pains and vomiting with or without fever. At the time of an epidemic every case of diarrhœa must be regarded as a "suspect," until the true nature of the symptoms is settled by bacteriological examination of the discharges. The stools and any linen that is soiled must be efficiently disinfected. 2. Serous Diarrhœa.—Diarrhœa becomes more urgent, with frequent large liquid stools, which presently assume the rice-water appearance. Or the attack may begin in this way without premonitory symptoms. There are griping pains in the abdomen and much bearing down, with great prostration. The tongue is covered with a thick, whitish fur and there is extreme thirst. In the course of a few hours vomiting occurs. Severe muscular cramps, especially in the legs and feet, add to the sufferings of the patient. Notwith-

standing the severity of the symptoms recovery may, in favorable cases, set in at this period. The pains and tenesmus may cease, the rice-water character of the discharges give place to stools that are fecal and bile-stained, the gastric irritability subside, and little by little the ability to retain water and nourishment return. 3. Stage of Collapse.—In other cases collapse symptoms rapidly develop. The appearance of the patient is due to the rapid withdrawal of fluid from the tissues. The skin is ashy gray, shrivelled, wrinkled and inelastic, and covered with a clammy perspiration; the features are shrunken, the eyeballs sunk in the sockets, the nose pinched, the cheeks hollow, and the surface cyanotic and mottled. The external temperature is subnormal but the internal registers 103°–104° F. (39.5°–40° C.) or higher. The pulse is feeble, thready, and uncountable. Diarrhœa frequently ceases and there is merely a continuous oozing of rice-water material from the anus. The voice is husky and whispering. The mental condition often remains singularly clear and alert. At the last, coma supervenes. This is the fatal stage of cholera. It lasts from a few hours to a day or two. The thin liquid stools are of a grayish-white color, resembling turbid whey or rice-water. They contain much granular matter and small whitish flakes of mucus. In other cases they are tinged with blood and have the appearance and odor of the washings of meat. They are alkaline in reaction, highly albuminous, and contain sodium chloride in large proportion. Under the microscope epithelial cells and bacteria, often comma bacilli in nearly pure culture, are seen. The blood in consequence of concentration shows a high leucocytosis. The urine is greatly diminished or anuria may be present. That which is voided is intensely albuminous. Microscopically it presents the characters of an acute parenchymatous nephritis. Saliva is scanty but the function of the sweat-glands is maintained. *Cholera Sicca.*—In rare instances the contents of the bowel are retained and collapse terminates in death without diarrhœa. 4. Reaction.—In the cases which survive the stage of collapse the symptoms characteristic of that condition gradually subside. The action of the heart grows stronger, warmth and color return to the skin, which regains its natural turgor, cyanosis disappears and is often replaced by a reddish mottling or erythematous blush, the stomach becomes retentive of water and bland fluids, the colicky pains and violent muscular cramps cease, the stools are much less frequent, and the secretion of urine is re-established. With these signs of improvement the disparity between the external and internal temperatures passes away. The patient now enters upon convalescence which is often protracted, but frequently interrupted by a relapse, which usually proves fatal.

Cholera-typhoid.—The stage of reaction may pass into a septic condition characterized by so-called typhoid symptoms, due to secondary infection. In some instances the predominant features are those of general sepsis, in others pulmonary, and frequently they are uræmic. Feeble, rapid pulse, dry tongue, muttering delirium, and stupor are followed by coma, which terminates in death. As in other epidemic diseases cases of every degree of severity occur.

Complications and Sequels.—The attack in the graver cases is so

severe and rapid in its course that complications as such are not common. Important sequels are inflammation of the mucous membranes, as diphtheroid colitis, pleurisy, and pneumonia, and abscess formation, especially parotid bubo. Muscle cramps may persist and subacute gastro-intestinal symptoms are often present for a long time.

Diagnosis.—Direct.—During an epidemic no doubt arises in well-developed cases. The clinical picture is unmistakable. The uncertainty in regard to first cases and suspects is usually quickly dispelled by the course of the attack. Bacteriological examinations are necessary. Special instruction in the examination of the stools should be arranged in the laboratories at the time of prevalence of cholera—microscopically, cultural methods, agglutination tests.

Differential.—*Cholera Nostras: Cholera Morbus.*—The symptoms and course of severe cases do not differ from those of Asiatic cholera. The cases are sporadic and occur in hot weather in temperate climates. There is often a history of improper food, chilling, or exposure. Vomiting, diarrhœa with rice-water stools, colic, muscular cramps, suppression of urine, cyanosis, and collapse may terminate fatally in the course of ten or twelve hours. The differential diagnosis can only be made by laboratory methods. *Arsenical and Other Poisoning.*—Vomiting, diarrhœa, and collapse are constant symptoms in acute poisoning by the preparations of arsenic, mercury, and the poisonous fungi. The absence of cholera, the sporadic occurrence of poison cases, the anamnesis, the evidences of the poisonous substance, or the vial or box in which it was contained, are important.

Prognosis.—The mildest cases recover; the severe cases almost invariably die. The mortality ranges in different epidemics between 30 and 80 per cent. In any given case alcoholism, old age, or diminished powers of resistance from other causes, marked cyanosis, a temperature much below normal, and early collapse are of ominous prognostic import.

XXII. BACILLARY DYSENTERY.

Definition.—A disease of the large intestine, usually acute but sometimes becoming chronic, occurring sporadically and in local epidemics, due to the bacillus dysenteriæ and characterized by tormina, tenesmus, and frequent discharges of mucus and blood.

Etiology.—Predisposing Influences.—Dysentery is a widely spread disease. It occurs in all parts of the world but is especially common in tropical and subtropical countries. While bacillary dysentery is much more prevalent and disastrous in hot climates, it is also common in temperate climates both as a sporadic and an epidemic affection. Overcrowding and neglect of sanitary requirements both in military and civil life are predisposing influences of great importance. It follows that dysentery constitutes one of the most serious difficulties in warfare, especially in tropical campaigns, and that its epidemic outbreak in overcrowded institutions not infrequently occurs.

Exciting Cause.—*Bacillus Dysenteriæ.*—Shiga, in 1898, discovered in the stools of these cases a bacillus, having specific characters, which he

regarded as the cause of the disease, and to which he gave this name. This organism has been found in the dysentery of the Philippines, Porto Rico, in that occurring in various points in the United States and Europe. It has been demonstrated in the summer diarrhœas of infancy.

There are several strains, as determined by the relative agglutinating power of the immune serum upon the bacilli in pure culture and the action of the bacilli upon various sugars, but the lesions produced are the same. Flexner's types are, (1) the Shiga, (2) the Flexner-Harris—the strain prevalent in the United States, and (3) Bacillus Y. The Bacillus dysenteriæ has never been isolated except from the stools or lesions in human beings. The mode of infection has not been demonstrated. The lesions comprise intense hyperæmia of the mucosa of the large intestine, with scattered points of hemorrhage, superficial necrosis over limited or extended surfaces, and enlargement of the solitary follicles. Deep ulceration is not present in the cases that are early fatal. In the most intense cases great thickening of the mucosa and other coats of the colon occurs, together with extensive necrosis and gangrene. The ileum may be involved.

Fig. 258.—Bacillus dysenteriæ (Shiga).

Symptoms.—The period of incubation does not exceed forty-eight hours. The onset is sudden, with abdominal pain and frequent discharges consisting at first of fæces, followed by mucus which soon becomes bloody. There is urgent inclination to go to stool, with twisting abdominal pain and violent rectal tenesmus. The pyrexia, moderate at first, soon rises to 103°–104° F. (39.5°–40° C.). Thirst is intense and there is complete loss of appetite. The pulse is rapid, small, and feeble. In the very severe cases the patient becomes delirious and death occurs in the course of the third or fourth day. In favorable cases the urgency of the intestinal symptoms gradually declines, the temperature falls, and convalescence may be fully established in three or four weeks. There are other cases in which, with subacute symptoms, the cases run a protracted course. Many of our soldiers return from the island possessions with chronic dysentery.

Among the important complications and sequels are malarial infection, subacute septic arthritis, pluerisy, pericarditis, endocarditis, and sepsis. Albuminuria, anæmia, œdema of the legs and feet, and various palsies due to neuritis occur in the protracted cases. In contrast to amœbic dysentery, abscess of the liver is extremely rare.

Diagnosis.—Direct.—Laboratory methods are necessary. In non-amœbic dysentery the B. dysenteriæ must be sought in the stools. It is isolated most conveniently from the shreds of mucus. In the acute cases the blood-serum agglutinates the bacillus in the Flexner-Harris strain in dilutions of 1-1000 up to 1-1500; the Shiga strain agglutinates less readily.

Differential.—Bacillary dysentery is to be distinguished from amœbic dysentery only by the methods of the laboratory.

Prognosis.— The outlook in the sporadic cases in temperate climates is favorable; in local epidemics less so, especially with bad sanitary arrangements. In active campaigns and tropical dysentery the death-rate is high. The dysentery of Japan has a mortality of about 25 per cent. After recovery there is very frequently prolonged ill health with gastro-intestinal symptoms and diarrhœa.

XXIII. THE PLAGUE.

Pestis; Bubonic Plague.

Definition.—An infectious febrile disease of the Orient, caused by Bacillus pestis, and characterized by glandular swellings or buboes, carbuncles, pneumonia, and, in many cases, hemorrhages beneath the skin and from the mucous surfaces.

Etiology.— Predisposing Influences.—This disease, the great pestilence of Europe and Great Britain for eleven hundred years, practically disappeared towards the close of the seventeenth century. It has not appeared as an epidemic in England since the Great Fire in London in 1666, the year following the Great Plague. Its cessation is due to modern methods of living. It has been said of Europe that when the shirt came in the plague went out. Always smouldering and frequently flaring up in the East, the plague occasionally slipped over into Lower Italy, Egypt, and other countries bordering on the Mediterranean, but not until the outbreak at Hong Kong in 1894 did it again threaten to become a world pest. Since that date it has continued its ravages, especially in India, where during the first six months of 1905 nearly 900,000 persons died of it, the highest mortality for a half-year made in the epidemic of eleven years' duration. During the past decade the plague has appeared in Egypt and other parts of the Mediterranean, South Africa, Oporto, Glasgow, New York, Mexican and South American ports, Australia and New South Wales. Occupation, age, and sex are without influence as predisposing factors. The disease spreads chiefly among the poorer classes. The prevalence is greatest in the hot season, but outbreaks sometimes occur during the coldest weather. Personal and household cleanliness are important. In Bombay few attendants upon the sick were attacked, and not a case occurred among the British soldiers engaged in police duty and disinfection. Only an occasional case occurs among Europeans living in infected regions.

Fig. 259.—Bacillus pestis. *a*, organism from culture; *b*, smear preparation from spleen.

Exciting Cause.—Bacillus pestis was discovered by Kitasato. It appears in the form of short, non-motile rods, with rounded ends, staining readily and more densely at the poles than in the middle, and decolorized by Gram's method. This organism has a characteristic growth in culture. It is present in the blood and lesions of the plague and in the dust of houses in which cases have occurred, and in the earth of the floors and adjacent parts. Dogs and cats and household vermin, as rats, mice, flies, and fleas, suffer from the infection, transmit it to others and to man, and die infected by its germ. Plague bacilli are present in enormous numbers in the hemorrhagic sputum in the pneumonic form of the disease, and may be found in the ordinary bubonic form.

Mode of Transmission.—The plague is not contagious in the ordinary sense. It is dependent on the disease in the rat, and is transmitted from rat to man by the rat flea. Cases commonly occur singly in a house. Multiple cases are usually simultaneous. Rat fleas are carried by personal and other fomites. Insanitary conditions except as to rats have no relation to plague.—Plague Commission, 1908.

Symptoms.—The period of incubation varies from two to five days. The following forms are described: 1. Rudimentary Form; Pestis Minor.—The patient is not in all cases ill enough to go to bed. There is moderate fever, with enlargement and tenderness of the inguinal glands, which sometimes undergo suppuration. These cases constitute a danger to the community by the presence of the bacilli in the discharges. 2. The Ordinary Form; Bubonic Plague.—The onset is sudden, with a chill which is immediately followed by fever, headache, backache, muscular soreness, great anxiety, and depression of spirits. The temperature progressively rises until the third or fourth day, when there is a more or less marked remission, followed by a further rise coincident with the development of the buboes, and accompanied by signs of septic infection, dry, brown tongue, delirium, stupor and collapse symptoms—secondary fever. Death frequently occurs at this stage. The swelling of the superficial lymph-nodes occurs between the third and fifth days, the inguinal nodes being involved in more than half the cases, less frequently those of the axilla or neck. The adenitis may undergo resolution, suppuration, or, in rare cases, gangrene, with the formation of deep sloughs. Necrosis of the subcutaneous tissue may give rise to more or less extensive carbuncles. Enlargement of the spleen occurs. Petechiæ are common, and the extensive subcutaneous hemorrhages that characterized the disease in certain epidemics gave to it in the Middle Ages the popular name of the Black Death. Hæmoptysis and other hemorrhages from the mucous surfaces have been especially noted in some outbreaks. 3. The Septic Form.—The symptoms are from the onset overwhelming, and death occurs in the course of three or four days without the appearance of buboes. Hemorrhages constitute a prominent feature. Metastatic abscesses are frequently found in the viscera. 4. The Pneumonic Form.—The disease may appear as a primary specific pneumonia, with the usual characters of infectious inflammation of the lungs. The type is bronchopneumonic, the fever high, the respirations rapid, the sputum hemorrhagic and laden with the bacilli.

The attack lasts only a few days and almost invariably terminates in death.

Diagnosis.—Direct.—Cases imported during the stage of incubation or the first cases in an outbreak may be readily overlooked. In suspected cases all uncertainty can be at once settled by a proper laboratory investigation. The bacteriological examination of the blood, pus from the buboes, the urine, and the sputum yields positive results. If necessary cultures should be made and inoculation experiments upon guinea-pigs. The danger of the importation of the disease at the present time renders an efficient inspection at the quarantine station at every port of entry imperatively necessary with bacteriological studies in the case of every suspect.

Prognosis.—Bubonic plague is the most fatal of the acute infectious epidemic diseases. In the larval forms recovery is the rule; in the septic and pneumonic forms death; in the ordinary bubonic form a great majority of the cases die. The statistics of the Middle Ages are unreliable but it is stated that the Black Death of the fourteenth century destroyed one-fourth of the population of Europe.

XXIV. MALTA FEVER.

Undulant Fever; Mediterranean Fever.

Definition.—An acute general infectious disease caused by the Micrococcus melitensis and characterized by irregular fever of long duration and remittent or intermittent type, with periods of apyrexia, by profuse sweating, rheumatoid pains, arthritis, and enlargement of the spleen.

Etiology.—Predisposing Influences.—The disease prevails widely upon the littoral and islands of the Mediterranean and is known as Mediterranean fever, rock fever, Neapolitan fever, Danubian fever. It has also been encountered in China and India, Manila and in the West Indies, and imported cases have been studied in this country. It is prevalent in summer as an endemic disease, not occasionally in circumscribed epidemics. It is not directly transmissible from the sick to the well. Malta fever is especially a disease of young adults. It has been particularly studied by the surgeons of the British Army stationed at Gibraltar and Malta.

Fig. 260.—Micrococcus melitensis.

Exciting Cause.—The Micrococcus melitensis was first isolated and studied by Brun in 1887. This organism is found in the circulating blood, and is present in the spleen during life and after death. It is pathogenic for monkeys, and cases of accidental infection in laboratory work have been reported. The serum of the patient after the fifth day causes agglutination of cultures of the organism in dilution of 1 to 10 or 1 to 50.

The milk supply at Malta is largely derived from goats, and Zammit, in 1905, made the important discovery that the goats of the island are infected with Micrococcus melitensis and isolated this organism from the milk and urine of those animals.

Symptoms.—The period of incubation lasts from six to ten days. The onset is preceded by prodromes not unlike those of enteric fever. The course of the disease is characterized by undulations of fever, 102°–104° F. (39°–40° C.), of distinctly remittent type, lasting as a rule from one to three weeks, and separated by intervals of incomplete or complete apyrexia of two or more days' duration. In rare cases during the pyrexial period the fever conforms to the intermittent type, without, however, manifesting the regular periodicity of the malarial infections. This irregular fever is indefinitely prolonged, lasting from three months, the average time,

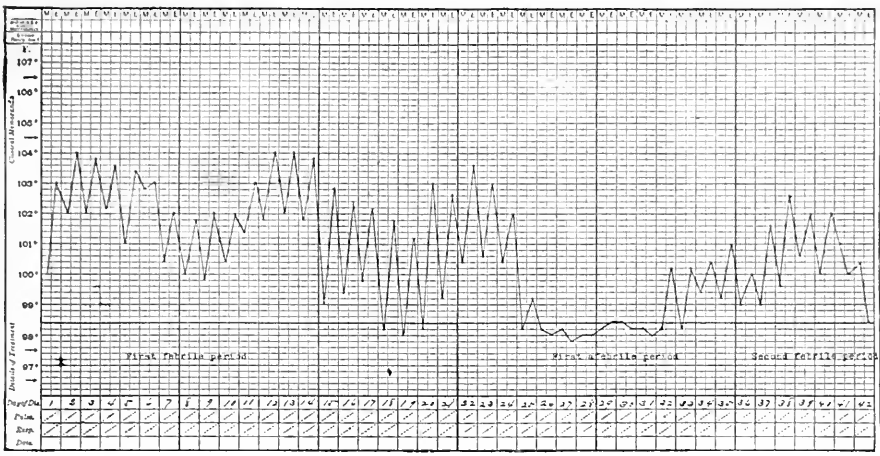


FIG. 261.—Malta or undulant fever.—Jackson. An average case may extend more than twice the duration shown on this chart.

to six months, and being in some instances prolonged by a series of relapses to two years. Obstinate constipation, progressive anæmia, and debility are common symptoms; the spleen is enlarged and tender; neuralgias, inflammation of the joints, with intra-articular effusion, painful inflammatory conditions of certain fibrous structures, and orchitis occur as complications. The wave-like range of the temperature has suggested the name "undulant fever." A malignant type, fatal in the course of a week or ten days, is recognized, and a so-called intermittent type with a daily rise of temperature toward evening has been described.

Diagnosis.—DIRECT.—In districts in which Malta fever is endemic the direct diagnosis is not attended with difficulty. It rests upon the character of the fever and its protracted course with intervals of apyrexia, the tendency to relapse, the headache, lassitude, and joint affection. Finally, the agglutination test is conclusive.

DIFFERENTIAL.—The true nature of the disease may not be apparent in imported cases. *Malaria* reveals itself upon an examination of the blood. The temperature is higher, the periodicity, as a rule, more dis-

tinctly defined. Joint pains are not prominent. The judicious use of quinine is curative—therapeutic test. *Enteric fever* differs from Malta fever in its temperature range and duration. If relapses occur they constitute a repetition of the primary attack and like it are attended by subcontinuous temperature, rose spots, and marked intestinal and nervous symptoms. A positive Widal reaction is conclusive. Endemic and epidemic influences are suggestive. *Dengue* is a pandemic disease and spreads with great rapidity. The joint affection is among the earliest and most conspicuous phenomena. The initial febrile paroxysm is of short duration and the recurrent paroxysm is characterized by a polymorphous rash. *Rheumatic fever* bears only a superficial resemblance.

Prognosis.—The mortality is about 2 per cent. Death is due to the debility resulting from indefinitely prolonged fever, the anæmia, or complications. Convalescence is hastened by change of climate.

XXV. TETANUS.

Lockjaw.

Definition.—An infectious disease caused by a bacillus—*B. tetani*—found in garden earth and in the excrement of herbivorous animals, especially the horse, characterized by tonic spasm of the muscles, with paroxysmal exacerbations.

Etiology.—Predisposing Influences.—Tetanus is essentially a wound infection—*tetanus traumaticus.* The view at one time entertained, that the disease may be cryptogenic—*tetanus idiopathicus*—or due to rheumatism or exposure to cold, *tetanus rheumaticus,* is no longer accepted. Many cases arise in consequence of insignificant wounds which are overlooked or entirely healed at the beginning of the attack. Internal injuries involving the mucous membrane, which elude observation, are to be considered in this connection. Whether or not infection may occur by inhalation of tetanus spores in the absence of a lesion of the buccal or respiratory mucous membrane is not known. Tetanus occurs in all latitudes but is much more common in tropical than in temperate climates, and among the colored than the white races. This is especially true of puerperal tetanus and tetanus of the new-born. In tropical America and the West Indian Islands tetanus has prevailed as a veritable public scourge.

Since the discovery of the tetanus bacillus and the diffusion of the knowledge of its saprophytic existence the prevalence of the disease has greatly diminished. Tetanus occurs more frequently in the summer and early winter than at other seasons of the year. It has been attributed to exposure to cold and to sleeping upon the damp ground. Aside from the frequency of tetanus among the new-born, age is without influence. The disease is said, however, to be less common after the sixteenth year. Sex is without influence except in so far as occupation renders males more liable than females. Occupation is an important predisposing influence. All those which involve liability to wounds of the extremities, with coincident fouling with earth, manure, or the excrement of animals, especially with that of the horse, or with dust or dirt containing such material, pre-

dispose to tetanus. Hence stablemen, teamsters, gardeners, and soldiers, especially cavalrymen, are particularly liable to the disease. In some military campaigns tetanus has contributed largely to the mortality.

The majority of cases occur after lacerated and crushed wounds, especially those involving large nerves. The disease is comparatively infrequent after incised wounds. The extent and severity of the wound has no direct relation to the liability to tetanus. The condition of the wound as regards its tendency to heal is entirely without influence; tetanus may occur when a wound has completely healed and a cicatrix has formed. The disease may follow the most trifling traumatism—the extraction of a tooth, the use of cupping glasses, the sting of an insect, the application of a blister. It is far more frequent after injuries of the extremities than in other parts of the body. The disease has been attributed to the use of the hypodermic syringe. At one time tetanus was common in hospitals and occasionally became epidemic in maternity institutions, a large proportion of the lying-in women and their children dying of this disease.

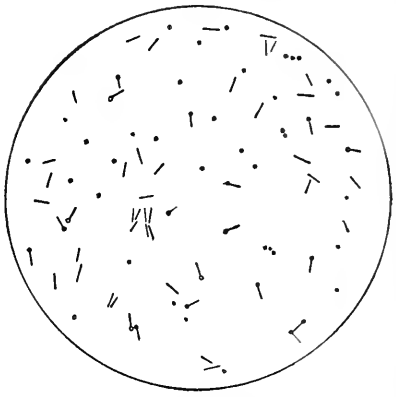


Fig. 262.—Bacillus tetani and free spores.

Diphtheria antitoxin, vaccine virus and bacterial vaccines, if not subjected to rigid control, may be contaminated with tetanus spores. Gelatin sometimes contains tetanus spores and ineffectually sterilized solutions of this substance used as a hæmostatic have caused human tetanus.

Exciting Cause.—The tetanus bacillus is characterized by a terminal spore, which gives it the appearance of a nail or pin. It is slightly motile, gram positive, anaërobic and pathogenic for animals. In wounds it multiplies, giving rise to an intensely virulent toxin, which extends by way of the nerves to the nerve centers and brain unless promptly neutralized by tetanus antitoxin. Its normal habitat is the intestinal tract of herbivorous animals. Its spores are characterized by great resistance. It has been found in the cultivated surface soil of all countries, but not beyond the depth of 30 cm. It has also been found in the dust of streets and in the woodwork of houses and furniture. It is frequently present in the excrement of animals and man. The comparative infrequency of tetanus is in strong contrast with the wide distribution of its cause.

Symptoms.—The period of incubation varies from three days to three weeks. In a majority of the cases acute symptoms manifest themselves between the fourth and the tenth day. In general the onset occurs between the eighth and fourteenth days, rarely later than the fourth week. Prodromes are infrequent. They consist of nausea, tenderness in the wound or scar, increased suppuration or spontaneous reopening of the wound, accompanied by restlessness and loss of sleep.

The attack is occasionally marked by shivering or an actual chill. The characteristic symptoms are continuous tension of the voluntary muscles, and the occurrence at irregular intervals of spasms of varying intensity. The tension and the spasms are commonly relaxed during sleep. In inoculated animals, these symptoms usually commence in the region of the wound and extend to other parts of the body. In man they often first involve the muscles of the neck and suggest an ordinary torticollis from cold. Tension and spasm of the masticatory muscles—*trismus*—soon occur and may be the earliest manifestations of the true condition. There is inability to open the mouth or protrude the tongue—*lockjaw*—and efforts to perform these actions provoke more or less persistent spasm of the facial muscles—*risus sardonicus.* Presently the abdomen is felt to be hard and board-like, as in the early stage of peritonitis, from contraction of the muscles. A sensation of oppression or pain in the precordia, extending to the spine, is frequently experienced, and has been attributed to spasm of the diaphragm. The pain at this time is not usually severe in proportion to the violence of the spasm; later it becomes more intense. In a short time the spasms extend to the voluntary muscles in all parts of the body and affect them with about equal severity. The dorsal and lumbar muscles may contract more violently than their antagonists, giving rise to *opisthotonos.* Forcible contraction of the abdominal muscles, causing *emprosthotonos,* or of the muscles of one side—*pleurothotonos*—are far less common and less marked. On the other hand, transient or persistent rigidity of the trunk and limbs—*orthotonos*—is frequently observed. The fingers are but slightly involved in the general stiffness and spasmodic contraction. The laryngeal muscles may be involved, causing noisy respiration, dyspnœa, or fatal asphyxia. The spasms may occur spontaneously and are apt to come on when the patient awakes from sleep. They are caused by trifling external impressions, as efforts to move, the arrangement of the bedclothing, or an examination of the pulse or heart. Attempt to swallow frequently brings on violent general spasm. Viscid saliva accumulates in the mouth and is swallowed with difficulty. Respiration is disturbed; there may be cyanosis; the expression is greatly changed. The spasms are followed by great exhaustion, with periods of quietude, drowsiness, and sleep. The duration of the paroxysms is variable. The intellect as a rule remains clear throughout the attack. In rare cases delirium has occurred. The pulse is increased in frequency during the paroxysms; in the intervals the pulse and respiration are commonly normal. The temperature is frequently normal until toward the close of the attack; in some instances there is fever from the onset, 104°–105° F. (40°–40.5° C.). A preagonistic rise is usual and often extreme, 106°–110° F. (41.1°–43.5° C.). Abundant perspirations occur during the paroxysms. The urine is decreased in quantity, concentrated, dark, and frequently albuminous. Its toxic coefficient is increased and its injection into animals has caused fatal tetanus. The bowels are constipated. The sphincters are contracted. The administration of a clyster may be difficult and catheterization impossible. These procedures are

not only hindered by the state of the muscles but they also cause violent general spasms.

The acute cases usually prove fatal about the third day. The milder cases may continue for two or three weeks or even longer. Recoveries usually take place in the prolonged cases. The disease is most severe and quickly fatal when it develops shortly after the injury. Death occurs in the acute cases, usually during the paroxysm, from asphyxia, œdema of the glottis, or cerebral hemorrhage; less frequently from sudden collapse or coma. In the prolonged cases it may be the result of exhaustion or of inhalation pneumonia. If the patient survives the fourth day the intensity of the symptoms may gradually diminish. Tetanus neonatorum is due to infection at the navel. The disease usually shows itself between the first and fifth days after the separation of the cord. The symptoms are the same as those of ordinary traumatic tetanus. The duration is variable, death commonly occurring about the third or fourth day. Recovery is rare.

The term puerperal tetanus is applied to those cases which develop in the lying-in woman. It frequently occurs as the result of abortion or from neglect of antiseptic precautions in confinement. Tetanus may occur after operations or injuries of the genitalia in non-pregnant women. Tetanus bacilli have been found in the uterus and vaginal discharges.

The Cephalic or Facial Tetanus of Rose originates from a lacerated wound in the region of the facial nerve and is characterized by trismus, difficulty in swallowing, and paralysis of the facial muscles upon the same side.

Diagnosis.—Direct.—This rests upon the occurrence of stiffness in the muscles of the neck, trismus, substernal pain extending to the back, followed by risus sardonicus, general tension of the muscles with paroxysms of spasms, and finally upon the bacteriological examination. The previous occurrence of trauma is most important. Courmont has shown that neither spontaneous nor experimental tetanus develops any agglutinating property in the blood.

Differential.—1. *Torticollis* following exposure to cold—so-called rheumatic torticollis. A history of trauma and the rapid development of the attack speedily settle any doubt. 2. *Trismus* associated with quinsy. Parotitis and local abscesses of the jaws or teeth may suggest tetanus, but a careful examination and the absence of general symptoms render the diagnosis clear. It is important in this connection to note that the rigidity of the masticatory muscles in tetanus is bilateral. 3. *Strychnia Poisoning.*—The spasms are very suggestive but trismus is rarely marked, and in the intervals between the paroxysms there is, in strychnia poisoning, no rigidity. 4. *Cerebrospinal Meningitis.*—Opisthotonos is a common and prominent symptom and in rare cases general spasms may occur, but the high fever and grave mental derangements stand in contrast to the absence of fever at the beginning and the mental clearness in tetanus. 5. *Tetany.*—Spasm of the extremities, the peculiar position of the wrists and hands and the feet and toes, the occurrence of the symptom described by Trousseau and that of Chvostek, together with the conditions under which the disease occurs, usually render the diagnosis a simple matter.

6. *Hydrophobia.*—The history of the case, showing the bite of an animal or man, the long period of incubation, the absence of trismus, the influence of attempts to swallow in producing the spasms, and the great restlessness and jactitation are of diagnostic importance. 7. *Hysteria.*—The paroxysms are occasionally attended by convulsions which suggest tetanus. The absence of antecedent trauma, the neurotic temperament, the interparoxysmal state, the emotional manifestations, the curious sensory disturbances, the existence of hysterogenetic areas render the differential diagnosis between this condition and tetanus easy.

Prognosis.—The worst days are the first four. The aphorism of Hippocrates still holds good: "The patient who survives the fourth day may recover." Traumatic tetanus is less frequent and less fatal in women than in men. The general mortality is about 80 per cent. The immediate prophylactic use of tetanus antitoxin serum in wounds defiled with manure, garden soil or any suspicious dirt, especially that of blank cartridges, and the radical treatment of penetrating wounds has greatly diminished the occurrence of cases. When symptoms have developed, the time for successful treatment is too often past. The later the disease shows itself after the occurrence of the wound of infection the milder is its course, and the more intense the initial symptoms the greater the danger. The mortality is greater in children than in adults. Restriction of the spasms to the muscles of the neck and jaw, and the absence of fever are favorable.

XXVI. HYDROPHOBIA.

Rabies; Lyssa.

Definition.—An acute infectious disease of warm-blooded animals, caused by an unknown specific virus contained in the saliva, characterized by convulsive and paralytic symptoms and communicated by inoculation to man.

The terms "hydrophobia" and "rabies" denote prominent symptoms. The Greek term "lyssa" is frequently used. The distinction between lyssa humana and lyssa animalis is unnecessary.

Etiology.—Predisposing Influences.—A distinction was formerly made between the hydrophobia of man and that of animals. Experimental medicine has established the fact that the disease in man and animals is the same. Climate is without influence. The disease occurs in all parts of Europe and is common in France, Holland, and England. At one time frequent in Germany, it has in recent years become rare as a result of the strict enforcement of laws regulating the muzzling and care of dogs. It is very common in Russia. In North America it is comparatively infrequent. The greater number of cases develop in the summer months, a fact which finds ready explanation in the out-door life and lighter clothing at this season. Men and children are affected more frequently than women who are less exposed and whose clothing affords some degree of protection.

Epidemics among animals can often be traced to a single case. The

spontaneous occurrence of the disease as the result of cold, heat, thirst, or other such cause has not been established. It is equally contrary to experience that hydrophobia arises spontaneously in man. Inoculation takes place in the vast majority of cases by the bites of animals suffering from the disease. Not every bite of an animal suffering from rabies produces the disease, since the saliva may be wiped off by the clothing. Especially dangerous are bites upon the face, hands, and the uncovered legs and feet.

Rabies occurs most frequently in the dog, wolf, and cat. Nearly all the domestic animals occasionally suffer as the result of inoculation. Rats and, in this country, the skunk are particularly liable. The disease is commonly propagated by the dog, which is on the one hand peculiarly susceptible and on the other hand liable to infect other animals and human beings, the disposition to bite being an especial manifestation of the attack. Next in importance in disseminating the disease is the cat. The propagation of hydrophobia by other animals is comparatively rare. Recent researches show that rabbits, guinea-pigs, and other warm-blooded animals in which hydrophobia was not formerly observed are susceptible.

Exciting Cause.—The nature of the virus is unknown. It is most abundant in the central nervous system and especially in the medulla oblongata. It is present also in the peripheral nerves though in much smaller amounts. The Negri bodies are round or oval structures found in the interior of the ganglion cells and having a capsule-like arrangement and vacuolar appearance. They are present in 90 per cent. of all infected animals. They are regarded by Negri as protozoa and the cause of the disease. These bodies do not pass through a Berkefeld filter. The virus of rabies is, however, filterable.

Noguchi (1913) cultivated the virus of rabies in material taken from the nervous system of infected rabbits and found after incubation granular, chromatic corpuscles of variable size, which he was able to grow for several generations. In the cultures he found also larger corpuscles which he regarded as identical with Negri bodies. Injected into laboratory animals these minute corpuscles gave rise to typical rabies.

Symptoms.—The period of incubation in man is irregular. Its average duration is from 20 to 60 days. It may, however, be prolonged to three months and exceptionally to six months.

The period of incubation is influenced by: (a) the susceptibility of the patient—it is shorter in children than in adults; (b) the animal communicating the infection. The incubation is shortest after the bite of the wolf and increases in the following order—the cat, the dog, and other animals. (c) The amount of the virus introduced—puncture wounds and extensive laceration wounds being followed by shorter periods; and (d) the part of the body upon which the inoculation takes place. Bites upon the face and head are followed by shorter periods of incubation, and next in order are bites upon the hands.

Stage of Prodromes.—There is irritation about the scar or the wound, together with pain. In some instances the healed wound reopens. Sensations of numbness may occur. Trembling and fibrillary contraction of the

muscles of the affected member have also been observed. Other prodromal symptoms consist of a feeling of depression, aching, and a sensation of pressure in the head. The patient becomes sad, loses interest in his surroundings, and prefers to be alone. He is unwilling to talk about the injury to which his illness is attributed. He is restless, apprehensive, and seeks relief from these symptoms in long solitary excursions. Children become depressed, lose interest in their ordinary play, and have irregular and disturbed sleep. There is increased general sensibility and bright light and noises are extremely distressing. There is laryngeal irritation which may show itself in slight hoarseness and occasional spasmodic cough. There may be difficulty in swallowing. Shivering may occur, which is followed by some elevation of temperature and increased pulse-frequency. Loss of appetite, nausea, and epigastric pain occur. This stage commonly lasts from two to eight days. It may however be much prolonged. In some instances the prodromal symptoms are altogether absent. Stage of Excitement.—There is an intense sensation of dyspnœa, interrupted by sighing and accompanied by a feeling of oppression and precordial distress. Difficulty in swallowing becomes more marked and it is commonly on account of these symptoms that the patient consults a physician. The difficulty in swallowing is characteristic. Attempts to drink or even to swallow the saliva produce violent painful reflex spasm of the muscles of the larynx and throat, which passes away to return with renewed intensity upon further attempts. So distressing is this symptom that the patient avoids taking fluid until compelled by the urgency of thirst. As the case progresses any irritation—a sound, a draught of air, the sight of water or the mere suggestion of it—may bring about the spasm. The superficial and deep reflexes are increased. The pupils are usually slightly dilated. This stage is characterized also by great excitability and restlessness. The patient is tormented by occasional hallucinations, his speech is short and broken, his voice hoarse, and during the convulsive attacks inarticulate sounds may be uttered which have been compared to the barking of a dog. In the maniacal excitement the patient sometimes bites himself. Very rarely, however, does he attempt to injure his attendants. The saliva is much increased. It is frothy and discolored and freely expectorated. At the beginning of the attack there is usually free perspiration. The urine is diminished and of high specific gravity. Albumin and casts are sometimes present. Sugar has been observed and in some cases hæmoglobinuria. Trembling of the tongue and hands is common and in some instances there is persistent tremor of the muscles of the face. Priapism occurs. The temperature is commonly elevated and may reach 100° to 103° F. (37.8°–39.5° C.). In many cases there is a preagonistic rise. The pulse is increased in frequency, irregular and intermittent. The respirations are irregular and shallow in the intervals between the convulsive paroxysms and sometimes assume the Cheyne-Stokes rhythm. The expression is anxious. Hallucinations and sometimes furious maniacal excitement attend the convulsive paroxysms. In the interval the mind is usually clear. Death may occur from asphyxia during the paroxysm. This stage

commonly last from 2 to 3 days. Paralytic Stage.—The spasms become less frequent and less severe. There gradually develops muscular relaxation. Difficulty in breathing disappears and it becomes possible to swallow. The patient sinks into unconsciousness, the heart's action becomes progressively more feeble, and death occurs in collapse. The paralysis may be limited to certain groups of muscles, as the tongue, the facial muscles, or the muscles of the eye. In other cases hemiplegia or paraplegia may occur. In rats as a rule, and occasionally in rabbits, the stage of excitement does not occur and the onset of the attack is marked by paralytic symptoms—*dumb rabies*. The disease occasionally assumes this form in human beings. It is apt to follow multiple and severe wounds inflicted by the rabid animal and has been attributed to proportionally large doses of the virus—an opinion supported by experimental investigations.

Diagnosis.—Direct.—There is nothing characteristic about the wound. It may heal promptly by first intention or by suppuration and granulation in the ordinary manner. The diagnosis depends upon the history of the bite of an animal suspected or known to be rabid, the occurrence of irritation in the wound or scar, the characteristic spasm of the muscles of deglutition and respiration, the intense reflex excitability, the short duration of the disease and the fatal issue. It is important in doubtful cases to determine by inoculation the presence or absence of the disease in the suspected animal. A small quantity of the substance of the central nervous system of a rabid animal inoculated subdurally will produce in a rabbit the paralytic form of rabies in the course of fifteen or twenty days.

Differential.—The following diseases may present superficial resemblances to hydrophobia in the human being: 1. *Hysteria*.—The reflex irritability may suggest hydrophobia, but the spasms lack early localization to the muscles of the throat and neck and the intensity of the later general convulsions. The stigmata of hysteria are present and the manifestations are indefinitely prolonged. 2. *Lyssophobia*.—The points of differential diagnosis are those which are to be considered in hysteria. The symptoms arise in hysterical and neurasthenic persons who have been bitten by dogs or other animals and who fear that they have been infected with hydrophobia. The pseudohydrophobic spasms are the result of autosuggestion. Cases of this kind are doubtless among the reported recoveries from hydrophobia. 3. *Tetanus*.—The incubation period is shorter; the convulsions are tonic; trismus is an early and persistent symptom, and the disease follows indifferent wounds and injuries rather than the bites of rabid animals. 4. *Various diseases—epilepsy, poisoning by datura stramonium, sunstroke, cerebral tumor, acute mania*—may present symptoms suggestive of hydrophobia and give rise to difficulties in diagnosis in cases in which there is the history of the bite of an animal suspected of rabies. The symptom-complex, course, and termination in these affections render the diagnosis as a rule a comparatively easy matter. 5. *Landry's paralysis* presents symptoms in some respects suggestive of dumb hydrophobia. The anamnesis is of great importance. Where there is a history of the bite of a rabid animal, or of an animal sup-

posed to be rabid, and the patient has been subjected to the Pasteur treatment, the differential diagnosis between the attenuated form of paralytic rabies, which occasionally develops subsequently, and Landry's paralysis is extremely difficult.

Prognosis.—Hydrophobia occurs in only 15 or 20 per cent. of individuals bitten by rabid animals. The danger is greatest when the wounds are lacerated or deep and especially when they involve the muscles. It is also greater in bites of the face and head than in those of the extemities. The probability that the patient may escape increases with the lapse of time. From the fourth month after the inoculation the danger rapidly decreases. According to Woodhead the mortality of patients bitten by rabid animals varied from 5 to 50 per cent. prior to the introduction of the Pasteur treatment, and the general mortality of those bitten by rabid animals was 16 per cent. Bollinger's statistics indicate that of patients bitten by dogs undoubtedly rabid 47 per cent. suffer and die from hydrophobia. Where the wounds have not been cauterized 83 per cent. of the cases succumb; where they have been cauterized 33 per cent. die. When the symptoms in the human being are well developed the prognosis is absolutely unfavorable, death taking place in a period varying from 12 hours to 4 days. Laveran, Roux, and others have reported cases in which some symptoms of hydrophobia have appeared in individuals undergoing the Pasteur treatment who have ultimately recovered. At the Pasteur Institute at Paris, of 27,719 cases of all kinds treated up to January, 1904, 117 died, a mortality of 0.42 per cent. These figures do not include a small number of cases in which the disease appeared during or within fifteen days after the treatments. Recovery occasionally takes place in animals inoculated in the laboratories.

XXVII. GLANDERS.

Farcy.

Definition.—An infectious disease of the horse caused by the Bacillus mallei and characterized by the development of nodules in the nose which undergo ulceration—glanders; nodules in the skin—farcy—and lymphangitis. It occurs occasionally in man as the result of accidental inoculation.

Etiology.—Predisposing Influences.—Among animals the ass is especially liable to glanders. Mules and horses are less so. Other animals may contract the disease. Wild animals in confinement, as in menageries, frequently develop it. Animals contract the affection usually through direct contact and commonly by the respiratory tract. Glanders in man occurs as a rule from exposure to the disease in horses. Stablemen, teamsters, coachmen, veterinary surgeons, and cavalrymen are especially liable. As a rule the infection is derived from chronic cases in the horse the nature of which for a long time remains obscure. Acute glanders in the horse is generally recognized and its further spread controlled by the destruction of the animal. The infection in man usually occurs through some more or less trifling lesion of the skin. Infection by way of the respiratory surfaces is

much less frequent. Infection may take place by way of the mucous membranes. The disease in a pregnant animal may be communicated to the fœtus.

Exciting Cause.—The specific germ—Bacillus mallei—morphologically resembles the tubercle bacillus but is shorter and thicker. It is chiefly communicated from the sick to the well by the discharges, including the urine and milk. When the bacilli are present in the circulating blood the course of the disease is very rapid. The patient perishes with acute symptoms.

Cases of glanders in animals have been reported in which recovery has followed the repeated injection of small but increasing doses of mallein, a substance isolated by Kalning and later by Hellman from cultures of the bacilli.

Symptoms.—For purposes of description it is convenient to make a distinction between glanders and farcy. They are due to the same cause and are very often associated. Acute and chronic forms occur. The period of incubation of acute glanders varies from three to five days. Prodromes are not uncommon, and consist of loss of appetite, nausea, and pain in the head and extremities. In acute glanders the *stage of invasion* is marked by general febrile disturbance. The mucous membrane of the nose is swollen and respiration interfered with. There is a scanty, bloody secretion which rapidly becomes more abundant and purulent. The nose is reddened and swollen. An erysipelatous inflammation may extend over the face. The nodules upon the nasal mucous membrane rapidly break down, with the development of extensive ulcers which may go on to necrosis. The lymph-nodes of the neck and the salivary glands are commonly much enlarged. Suppurative lesions of the skin, lymphangitis, and an inflammatory enlargement of the superficial lymph-nodes occur. *Stage of Eruption.*—The eruption is sometimes abundant, especially on the face and extremities, particularly the larger joints. It has been mistaken for variola. Only exceptionally do the pustules show umbilication. In some instances they are confluent. There is troublesome cough with abundant sanguinolent or mucopurulent foul-smelling expectoration. Suppurative arthritis may occur. Hemorrhages into the skin and mucous membranes have been noted. Colliquative sweating, diarrhœa, stupor, convulsions, and coma are followed by death, which usually takes place in the course of the second or third week of the attack. When an acute attack develops in the course of chronic glanders death may occur as early as the second or third day. (Fig. 264.)

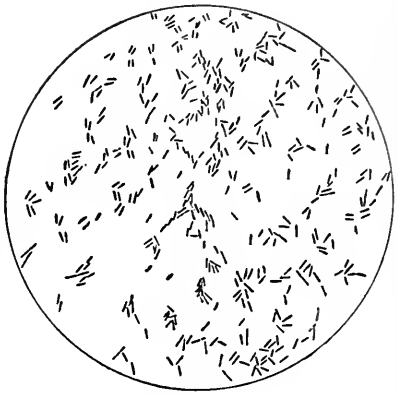


Fig. 263.—Bacillus mallei.

Acute Farcy.—The infection in man usually takes place from inoculation by way of the skin. There is intense inflammatory reaction with phlegmon formation which rapidly breaks down into an ulcer with irregular,

abrupt edges, from which extend painful reddened lines marking the course of the lymph vessels. The corresponding lymphatic glands are swollen, tender, and painful. The swollen lymphatics are known among veterinarians as *farcy-pipes;* the nodular dilatations in their course as *farcy-buds* or *buttons.* If the lesion be situated upon one of the extremities the limb rapidly becomes œdematous. Phlebitis may occur and abscesses form in the subcutaneous connective tissue. In other cases there are no signs of local inoculation. The sickness begins with the constitutional symptoms characteristic of glanders. In the course of from three to seven days small nodules occur in distant parts of the body which rapidly undergo suppuration with the formation of deep ulcers and areas of gangrene. The joints

Fig. 264.—The pustular eruption of glanders, often mistaken for small-pox. (Bovaird.)

may be involved and abscesses form in the muscles. The constitutional symptoms are those of an acute infection. The attack frequently begins with a chill or shivering. The fever is constant and may be intense. It does not conform to type. Remissions and intermissions occur. The mucous membrane of the nose may not be involved and the eruption may be absent. In the acute cases the bacilli have been found in the urine, both in animals and man. The termination is commonly in death in the course of the second week.

Chronic Forms.—The disease develops insidiously. Fever as a rule is absent. If the infection takes place through a lesion of the skin, similar manifestations to those in acute farcy may occur, developing however more slowly, and only after some time do symptoms of glanders or farcy appear. Symptoms referable to the organs of respiration are prominent. They consist of sensations of fulness in the nose, hoarseness, cough, an

abundant nasal secretion, and later dark, dry crusts. Upon examination catarrhal inflammation and ulceration are discovered. The condition is often looked upon as a chronic nasal catarrh. The process may last for months. In some cases recovery has taken place. More commonly the acute form of the disease develops or the patient dies of exhaustion. In chronic farcy the patients experience for some weeks pains in the limbs and joints. At the end of this time subcutaneous nodules develop. These undergo suppuration and form more or less extensive abscesses and ulcers. In some instances they show a tendency to heal; in others healing may take place and the scars after a time break down. The lymph-nodes are not often inflamed and the eruption is rare. Chronic farcy may last, with periods of rest and recrudescence, for two or three years and end in recovery. Most of the cases, however, terminate fatally with acute symptoms.

Diagnosis. — Direct. — The diagnosis of glanders or farcy depends upon the occurrence of the foregoing symptom-complex. With a clear history of the case and a knowledge of the occupation of the patient the diagnosis in acute cases is not difficult. In chronic cases no suspicion of the true nature of the disease may be entertained. A positive diagnosis can be reached by bacteriologic methods. Strauss recommends for diagnostic purposes the injection of cultures of the secretion into the peritoneal cavity of a male guinea-pig. After two days, in positive cases, there develop swelling of the testicles and granular inflammation of the tunica vaginalis; later a specific orchitis, which undergoes suppuration. The animal dies in the course of two or three weeks and the visceral lesions of glanders are found. Mallein is frequently used for diagnostic purposes.

Differential.—In the beginning of the acute cases the symptoms and course of the temperature may suggest enteric fever, and the joint pains rheumatic fever. Later glanders is to be distinguished from erysipelas and pyæmia. The urgency of the symptoms, the well-defined local manifestations, and the course of the attack will usually render the diagnosis a comparatively simple matter. The indolent serpiginous ulcers of chronic farcy may suggest tuberculosis or syphilis.

Prognosis.—The prognosis is in a high degree unfavorable. Recovery takes place very rarely in the acute cases. Chronic glanders usually terminates in death. In chronic farcy, recovery occurs in about 50 per cent. of the cases.

XXVIII. ACTINOMYCOSIS.

Definition.—A chronic infectious disease caused by the Streptothrix actinomyces or ray fungus, characterized by granulomatous new formations and multiple abscesses, in the pus of which are found peculiar bodies containing the organisms.

Etiology. — Predisposing Influences. — This disease occurs in all parts of the world. Those cereals armed with stiff or thorny processes may serve as carriers of the fungus. Barley and rye may be especially named. Cattle are most exposed to the danger of infection at the time of the second dentition and in the autumn and winter. Low and damp localities favor the infection. The fungus penetrates the tissues by way of pre-existent lesions of the mucous membranes or through wounds inflicted by the

spears of grain or pointed straws. The usual region of infection both in man and animals is the mouth; less commonly the gastro-intestinal canal, the lungs, or the wounded or abraded skin. The infection may be acquired by drinking water contaminated by the discharges from the mouth of an animal suffering from the disease. There is no reason to believe that infection occurs by means of the milk or flesh of diseased animals. Cases have occurred at all ages, from five to seventy years. Men suffer more frequently than women in the proportion of 5 to 3. Those occupations which involve habitual contact with cattle and their food must be regarded as predisposing causes.

Exciting Cause.—The parasite has been variously classified. Israel and Boström described it as a cladothrix; more recently it has been regarded as belonging to the streptothrix group. It appears in the pus as minute specks, which are yellowish or brownish by reflected light—sulphur granules—and often greenish by transmitted light. These granules vary in diameter from one-half to two millimetres and consist of a central core of filaments among which are cocci in varying numbers surrounded by a mass of radiating filaments, many of which present bulbous or clubbed extremities. The earliest developmental forms consist of smaller granules of a gray color and translucent appearance composed of a thick mass of threads either single or branched, closely interwoven at the centre and possessing the ray-like arrangement. The organism is polymorphous. In animals the club-shaped forms are more common; in man the filamentous. Both threads and clubs are present in cases in which the process is active. Ordinary pyogenic bacteria are present in varying numbers. The ray fungus has been grown upon artificial culture media and actinomycosis has been successfully inoculated both directly and by the artificially grown organism.

Fig. 265.—Streptothrix actinomyces.

Symptoms.—Actinomycosis is at first a local disease. Its course is generally chronic, and as distant organs become involved it presents the clinical picture of a chronic pyæmia. In very rare cases rapid dissemination may occur by way of the blood-vessels, and the disease run an acute course.

Gastro-intestinal Form.—The infection takes place by way of lesions in the mucous membrane of the mouth or throat or through the tonsils. The jaw is very commonly involved in cattle, much less frequently in man. There is swelling of the side of the face, usually involving the lower, rarely the upper jaw. The appearance may suggest sarcoma or a phlegmon. Sinuses form and the characteristic pus is discharged. Burrowing may take place in various directions. Indolent ulcers are common. The duration is variable. Very rarely the fatal issue occurs as the result of secondary infection or embolism in a few weeks. The usual course is chronic and may extend over years. The tongue may be involved either

primarily or secondarily. One or more circumscribed nodules form and in the course of a few weeks undergo softening and may be incised. Intestinal actinomycosis commonly involves the region about the cæcum and the appendix, or the sigmoid flexure and the rectum. Metastases are common. Pericæcal abscesses have been reported. The anus may be involved. Actinomyces have been found in the stools. Peritonitis is a common termination, but the disease may run a very chronic course with septic phenomena and cachexia.

Respiratory Form.—Actinomycosis of the neck may directly involve the larynx or may give rise to laryngeal œdema. The lungs may be involved primarily or secondarily. The lesions are less characteristic. In many cases they are merely those of a chronic bronchial catarrh. In others the tissue of the lungs is studded with gray nodules, resembling miliary tubercles and consisting of granulation tissue surrounding masses of the parasitic growth. In other cases the lesions are those of chronic bronchopneumonia with interstitial changes and a tendency to softening and the formation of cavities. As the process advances it involves the pleura, which may become adherent and greatly thickened or undergo suppurative changes leading to circumscribed empyema. Fistulous tracts are formed which open at the inner border of the scapula or elsewhere along the spinal column. Erosion of the vertebræ and necrosis of the ribs and sternum may occur. The clinical phenomena are those of pulmonary tuberculosis or fetid bronchitis. Actinomycotic granules are not always present in the sputa. As the disease advances there are septic symptoms with progressive emaciation and night-sweats. In rare instances the condition may simulate enteric fever. The duration varies from a few weeks to two or three years. Recovery is rare.

Cutaneous Form.—Cutaneous actinomycosis is very rare. It appears in the form of circumscribed tumors of a mottled purplish red and yellow color, varying in diameter from 1 to 3 or 4 centimetres, presenting one or more crater-like ulcerative openings, from which is discharged a clear sticky fluid sometimes containing the characteristic granules. In some instances the ulcerative process, while undergoing cicatrization at the centre, advances at the periphery. The condition is chronic and intractable.

In some few instances other regions have been primarily involved, especially the reproductive organs in the female, and the orbit. Bollinger reported a case of primary disease of the brain. In the other recorded cases the cerebral lesions have been the result of metastasis. The symptoms are those of cerebral tumor or abscess.

Diagnosis.—Direct.—This rests upon the presence of the actinomyces in the pus. Local tumor formation with a tendency to implication of bone and formation of multiple sinuses should arouse suspicion. Visceral actinomycosis gives rise to obscure symptoms. Tumors involving the lower jaw and the neck with multiple fistulæ are very suggestive. In the examination of the sputum some forms of degenerate epithelium and the Leptothrix buccalis may present strong points of resemblance to detached threads of the ray fungus.

Differential.—Actinomycosis of the lungs may resemble forms of chronic bronchitis and tuberculosis. Tuberculosis of the gastro-intestinal

tract may give rise to local peritonitis, infiltrations, abscess formations, and fistulæ, which cannot in the absence of the actinomyces be distinguished from similar conditions due to other causes. Cutaneous actinomycosis may resemble lupus and the lesions in the tongue may be mistaken for carcinoma, cysts, or syphilitic gummata.

Mycetoma or Madura Foot. — This curious disease of hot climates presents points of resemblance to actinomycosis. It is a chronic destructive local inflammation of the foot, or more rarely of the hand, resulting in an excessive proliferation of connective tissue. There are two varieties of the disease: the pale or ochroid form which is characterized by yellowish-white or brownish granules in the discharge, and the melanoid form which is characterized by dark brown or black masses of varying size. The disease shows no tendency to formation of visceral deposits. It was early described by Van Dyke Carter as a fungus disease. An organism has been cultivated from the pale variety which has been thought to be closely related but not identical with actinomyces.

XXIX. ANTHRAX.

Wool-Sorter's Disease; Malignant Pustule.

Definition. — An acute, infectious, epidemic disease of vertebrate animals, particularly sheep and cattle, caused by the Bacillus anthracis, and occurring sporadically in man as the result of accidental inoculation.

Etiology.—Predisposing Influences.—Anthrax is readily communicated from the domestic animals to man. Those occupations which involve direct or indirect contact with living or dead animals suffering from the disease constitute the chief predisposing cause. Individuals especially liable may be grouped as follows: 1. Farmers, shepherds, drovers, farriers, and veterinary surgeons. 2. Slaughterers and butchers. 3. Tanners, skin dressers, rag sorters, and workers in wool, hair, and horn. 4. Those who come in contact with persons following the foregoing occupations or who live in their neighborhood. 5. Anthrax may be transmitted from one person to another, and is under certain circumstances communicable from the human dead body to those coming into contact with it.

Anthrax is the most widely spread and destructive of the epizootics. All vertebrate animals are susceptible to anthrax, the herbivora being most liable, the omnivora less so, and carnivora only under unusual circumstances.

Exciting Cause — The Bacillus anthracis. This organism usually finds access to animals by way of the gastro-intestinal tract from infected fodder or infected pastures or water. Pasteur held that the earth-worm plays an important part in bringing to the surface and distributing bacilli from the buried carcasses of infected animals. Certain localities thus become permanently infected. The disease is directly inoculable and the infection may take place by the bites or stings of insects. Omnivorous animals, as the hog, dog, cat, and rat, though less susceptible, sometimes contract the disease by feeding upon infected carcasses. The disease does not spread by mere contact or association. The danger of infection

is greatly diminished if the carcasses of animals dead of the disease are buried unopened. Occasionally local outbreaks of anthrax among cattle, sheep, and other animals, in regions in which the disease does not prevail continuously, have been traced to imported hides, wools, and hair. These, not being thoroughly disinfected, are washed, the water being discharged into streams and sewers. In some instances the refuse from the manufacture of such articles is utilized for manure, and farms and fields thus become infected.

Fig. 266.—Bacillus anthracis.

Symptoms.—The cases may be grouped, according to the seat of the primary lesion by which the infection takes place, into (a) external or cutaneous anthrax, and (b) internal or visceral anthrax, of which there are pulmonary and intestinal forms.

(a) **External or Cutaneous Anthrax.**—1. Malignant Pustule or Vesicle.—The term "malignant pustule" is inappropriate and misleading. The condition is in some cases not malignant and the lesion does not suppurate. Anthrax is literally a burning coal. The general condition is known as anthracæmia. The term *charbon*—coal—is applied by the French to the local lesion of the skin, and *fièvre charbonneuse* to the general disease. The inoculation almost always occurs on some exposed part, as the arm, face, neck, or chest. The period of incubation varies from a few hours to two or three days. The early symptoms are local irritation and itching. A papule forms which rapidly becomes vesicular. There is surrounding redness and considerable brawny swelling. By the third day the vesicle ruptures, leaving a brown base exuding serum. In the course of twenty-four hours a black, dry, depressed eschar forms, around which at a little distance are several small secondary vesicles, sometimes discrete, sometimes confluent. The œdema extends for some distance and is very tense and deep. The related lymph-nodes are swollen and tender. Lesions upon the face or neck cause extraordinary swelling and disfigurement. Implication of the larynx and mediastinal glands gives rise to great difficulty in breathing and swallowing. Pus does not occur in favorable cases until the eschar begins to separate, usually toward

Fig. 267.—Anthrax pustule; early stage.—Royer.

the end of the second week: The severity of the general symptoms has no constant relation to the amount of local disease. Cases with marked local lesions may show but slight constitutional disturbance. Commonly symptoms of general infection rapidly follow the appearance of the papule, or they may be deferred for some days. There is a feeling of illness, chilliness, thirst, vomiting, and restlessness. In many of the cases the symptoms are those of the internal affection. Death may take place in from three to five days. In favorable cases the constitutional symptoms are slight, the eschar suppurates, and the wound heals. 2. Malignant Anthrax Œdema.—Swelling appears in the eyelids or elsewhere on the head, hands, and arms. Neither papule nor vesicle develops and there is no characteristic eschar. The œdema may be very extensive and occasionally follows the constitutional symptoms. Extensive areas of gangrene may result, with grave constitutional symptoms. A remarkable characteristic of the external forms of anthrax is the mental condition of the patient. With the gravest symptoms the mind may be perfectly clear and the patient manifest no indications of anxiety or distress up to the time of death.

Fig. 268.—Anthrax—fifth day; œdema of neck and thorax. —Royer.

(b) **Internal or Visceral Anthrax.** — 1. Pulmonary Anthrax (*Wool-sorter's Disease; Anthracœmia*). — This form of anthrax occasionally develops in those exposed by their occupations to the inhalation of anthrax spores in dust arising from the products of diseased animals. Wool and hair imported from Russia, Asia, Egypt, and South America appear to have been the cause of the disease in a large proportion of the cases. The symptoms are often indefinite until the approach of death. Prodromes are not common. The onset is usually acute. The patient suddenly feels out of sorts, has shivering, chilliness, uneasiness about the chest and stomach, and sensations of great weakness and weariness. In the course of a day or two, without having expressed sensations of being seriously ill, the patient may fall into a condition of collapse which terminates a few hours later in death. The tongue is moist and coated, thirst is moderate, and there may be weight and uneasiness at the stomach with complete loss of appetite. Vomiting and diarrhœa also occur. Symptoms referable to the respira-

tory system consist of a feeling of oppression, quickened respiration, cough, not commonly severe, with or without expectoration. The pulse is usually weak and rapid, out of proportion to the severity of the other symptoms, and toward the close of the case becomes irregular and uncountable. The heart sounds are greatly enfeebled. Wandering or active delirium, convulsions, and coma have been observed. The skin is moist. The temperature rises to 102°–103° F. (38.9°–39.5° C.) and may reach 105°–106° F. (40.5°–41.1° C.). It is commonly four or five degrees higher in the rectum than in the axilla. The urine is scanty, dark-colored, and of high specific gravity. Albuminuria is common. 2. Intestinal Anthrax (*Mycosis Intestinalis*).—This form is rare in man. Infection occurs by way of the stomach and intestines in consequence of eating the flesh or drinking the milk of diseased animals. The symptoms are those of intense poisoning, with gastro-intestinal irritation, and consist of nausea, persistent vomiting, abdominal pain, and diarrhœa. There is great weakness, restlessness, and difficulty in breathing. The pulse is small and rapid, the surface of the skin cold and moist, the face and extremities are slightly cyanotic. The rectal temperature is but slightly above normal. Hemorrhage from the mucous surfaces may occur and is sometimes accompanied by petechiæ and cutaneous abscesses. The spleen is enlarged. The blood is dark and fluid and contains the bacilli. Convulsions and coma are followed by collapse, and death occurs in from two to seven days. Instances have been recorded of local outbreaks in which the symptoms have developed at about the same time in a number of individuals.

The external form of anthrax may be associated with both the pulmonary and intestinal forms of the disease. Eppinger has shown that rag-picker's disease is a local anthrax of the lung and pleura with general infection, and a consideration of the pathological anatomy justifies the conclusion that the intestinal form also begins as a local process to which the constitutional symptoms are secondary.

Diagnosis.—Direct.—In both the external and internal forms of anthrax the occupation of the patient is of diagnostic importance. In external anthrax the direct diagnosis rests upon the character of the papule on an uncovered portion of the body, the rapid development of a vesicle, the redness and extensive brawny induration extending along the lymphatics to the neighboring glands. Microscopical examination of the contents of the vesicle may show the presence of anthrax bacilli. Cultures and inoculation experiments in a guinea-pig or white mouse give conclusive results in the course of two days, the animal dying and the internal organs showing anthrax bacilli in enormous numbers. These organisms may not appear in the blood until shortly before death. The appearance of the local lesion upon the third or fourth day is very characteristic. The central depressed eschar, the surrounding vesicles, redness, extensive œdema, with comparatively little pain, are significant.

Differential.—An ordinary *boil* or *carbuncle*. This rests upon the absence of suppuration and of a moist yellow slough. *Phlegmonous Erysipelas and Cellulitis.*—Anthrax may be distinguished by the absence of pain and of marginal secondary vesicles in the case of slough. *Chancre.*—The differential diagnosis rests principally upon the rapidity of the progress

and more serious constitutional symptoms of anthrax. *Glanders.*—There is an absence of the profuse purulent discharge from the nostrils. The direct diagnosis of pulmonary anthrax in the early stage is impossible. Later the gravity of the illness in connection with the symptoms above described, in an individual exposed to infection in his occupation, is highly suggestive. The direct diagnosis can, however, be made in some instances by microscopical examination of the blood. If this be negative inoculation experiments should be performed. The progress of the case in intestinal anthrax is so rapid, and the symptoms so closely resemble those of gastro-intestinal poisoning due to other causes, that a positive diagnosis during life is usually impossible.

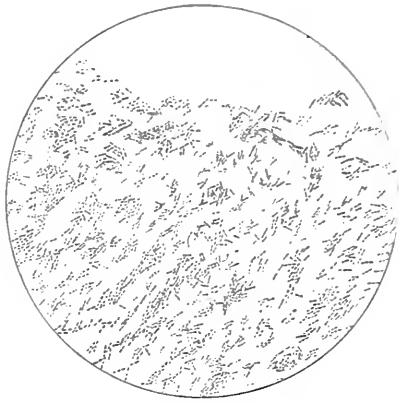
Prognosis.—Every case of anthrax may be regarded as a grave illness, but cases of spontaneous recovery are not altogether uncommon. The mild cases are most frequent in children and the intensity of the attack in man is said to correspond to the intensity of the disease in the animal from which the infection is derived. The prognosis is much more favorable in localized external anthrax than in the internal form. Malignant œdema of the face or neck is dangerous to life, partly by its extent and partly through the pressure exerted upon the structures of the neck, especially of the great vessels. The prognosis in anthrax œdema is by far graver than that of malignant pustule. Inhalation anthrax—the *rag-picker's disease*—gives a mortality of 50 per cent. The graver cases with severe fever, rapid prostration, and the evidences of extensive pulmonary inflammation terminate in death. Bell states that "no case demonstrated during life to be intestinal anthrax has ended in recovery."

XXX. LEPROSY.

Lepra; Elephantiasis Græcorum.

Definition. — A chronic, infectious, endemic disease caused by the Bacillus lepræ, characterized by a disseminated nodular infiltrate in the skin and mucous membranes—tubercular leprosy—or lesions of the nerves —anæsthetic leprosy. In the complete or generalized disease both sets of lesions are present—the mixed form.

Etiology.—Predisposing Influences.—No race is exempt. Leprosy occurs in all latitudes, in moist and dry climates, alike at the sea level and in mountainous settlements. Congenital leprosy is very rare; in fact its occurrence is doubted. The disease sometimes shows itself in childhood, but the vast majority of the cases develop in early adult life. In some instances it has first appeared in extreme old age. Males are affected in greater proportion than females. The mode of life is not without influence. The poor suffer more frequently than the well-to-do, but the latter do not escape. It has been thought that the habitual or exclusive use of certain articles of diet, as vegetables, salted food, food without salt, fish or pork, predispose to leprosy, either by the ingestion of the bacilli or by rendering the tissues less resistant to their development. Leprosy prevails in mountainous districts, as Kurdistan and Kashmir, where a fish diet is unknown, and among the Brahmins



who never taste fish. Furthermore systematic examinations of fish and preparations of fish in countries in which the disease is endemic have failed to reveal the presence of the Bacillus leprae.

The Exciting Cause.—The Bacillus leprae constitutes the specific infecting agent. Nothing is known of the distribution of the Bacillus leprae outside of the human body. It has not been found in the tanks in which lepers bathe, nor in the soil about the graves of lepers, although in some few instances it has been discovered in the soil of the paths and banks surrounding asylums. It is a slender, acid-fast, non-motile organism present in great numbers and arranged in a peculiar parallel manner in the cells and lymph spaces of the lesions. They are commonly present in the nasal secretion and sometimes present in the saliva, sputum, fæces and the blood of infected persons. They have been found in the milk of nursing women. Attempts at animal and human inoculation have yielded unsatisfactory results. This fact may be explained perhaps by the great length of the incubation period. In the case of Keanu, a Hawaiian criminal, pardoned in consideration of inoculation, who became leprous two years later, the experiment was vitiated by the fact that relatives were subsequently ascertained to have been leprous.

The Wassermann reaction and the complement fixation for tuberculin have been found to be positive in a large proportion of lepers.

It has been asserted that vaccination may be the means of transmitting leprosy. The danger cannot arise where bovine vaccine is used. The majority of lepers have never been vaccinated, and in countries where leprosy is steadily diminishing vaccination is becoming more general.

Until recently a belief in the hereditary transmission of leprosy was generally entertained. The present drift of opinion is against this view. The transmission of the disease takes place under conditions that are not well understood. When leprosy is carried by immigrants into highly civilized countries it rarely spreads. In countries where the disease has been endemic its diffusion is largely influenced by the degree of association of the lepers with the healthy. When the intercourse with lepers is controlled by legal enactments or a general dread of the disease, its prevalence is circumscribed and limited. The transmission of the disease by the conjugal relation is rare. Physicians in charge of hospitals and asylums for lepers rarely contract the disease. From 9 to 10 per cent. of the helpers in the leper settlement at Molokai have developed the malady. Prolonged exemption does not indicate permanent immunity.

Symptoms.—(a) Tubercular Leprosy.—The period of incubation varies from a few months to several years. The prodromal symptoms consist of irregular fever, weakness and prostration, loss of appetite, and impaired nutrition. Repeated epistaxis is not uncommon. After a time areas of cutaneous erythema appear. These may be sharply defined and in some instances are anæsthetic. Later they undergo pigmentation. These spots vary in size and some of them may disappear. After a time pea-sized or larger nodules appear which may run together and form large tuberculous masses. These tubercles are at first soft and elastic and slightly tender upon pressure; later they become firmer and are insensi-

tive. They may develop upon any portion of the body. The scalp is, however, usually exempt. They are most common upon the face, the dorsal surfaces of the hands and feet, upon the ankles, wrists, and forearms, and the outer aspect of the thighs.

The lesions progressively involve new areas of skin with the formation of fresh tubercular masses, and as the older ones undergo ulceration areas of cicatrization form. These changes in the skin undergo their most marked development upon the face, producing the characteristic *facies leonina*. The superficial lymphatics generally become enlarged early in the course of the disease. The eyelashes and eyebrows fall out and there is atrophy and loss of hair elsewhere upon the body. The hairy scalp is usually unaffected. The mucous membrane of the upper air-

FIG. 270.—Early stage of tubercular leprosy. —German Hospital.

FIG. 271.—Tubercular leprosy.

passages undergoes infiltration, with the formation of tubercles. These speedily break down, giving rise to painful ulceration. Changes in the voice occur. It becomes harsh or nasal, or more or less complete aphonia may develop.

As cicatrization takes place various deformities result, as stenosis of the mouth or palate, and laryngeal stenosis. The process involves the eyelids and extends to the ocular conjunctiva and the cornea, resulting in ultimate destruction of vision in from 66 to 75 per cent. of the cases.

The duration of the disease is indefinite. The patient may live for years, becoming more and more deformed and helpless. Death commonly results from exhaustion, colliquative diarrhœa, or inhalation pneumonia; sometimes from stenosis of the larynx or trachea or laryngeal œdema.

(b) ANÆSTHETIC LEPROSY.—The period of incubation is commonly more prolonged than in the tubercular form. The onset is insidious and

characterized by subjective cutaneous symptoms, as hyperæsthesia, pruritus, and pain. Fever is not common. Persistent and troublesome pains in the limbs are frequent. Hyperidrosis may be an early symptom. Trophic disturbances may give rise to the formation of bullæ. Patches of erythema varying in size from one to several centimetres, usually circular or oval in outline, form upon the trunk and limbs. After a time these spots disappear, leaving areas of anæsthesia, but anæsthetic patches may occur without the development of maculæ. The erythematous spots show a variety of tints, from pinkish-red to a bluish- or brownish-red color, and many of them undergo pigmentation. As the areas become anæsthetic the pigment may gradually disappear, leaving well-defined white or yellowish patches in striking contrast to the surrounding skin. The superficial nerve-trunks are felt to be enlarged and nodular. The hair of the affected surfaces may become white or fall out, and as the disease develops there is complete suppression of perspiration. Similar lesions may appear upon the face. They may remain discrete or become confluent. As the disease advances they are frequently the seat of bullæ, some of which undergo involution, with cicatrization, others break down, forming more or less superficial ulcers, which on healing leave conspicuous scars, which are at first dark but later become pale, smooth, and shining.

Fig. 272.—Anæsthetic leprosy.

The modifications of sensibility consist first of exaltations of sensibility, such as have been described; second, of perversions of sensation, which consist of dysæsthesias, formication, numbness, and delayed sensation; and third, abolition of sensation, which is more or less complete. The trophic changes involve the conjunctivæ and the mucous membranes of the nose, mouth, and throat, which may become dry and red and the seat of areas of superficial ulceration. The ulcers which form in the anæsthetic patches developing in the hands and feet may be very destructive, giving rise to contracture and necrosis, which produce distressing deformities, the loss of fingers and toes, and the development of perforating ulcers. Spontaneous resorption of bone may take place.

In favorable cases the disease may last for a long period without the development of marked trophoneurotic changes. The average duration of life in this form of leprosy is about twenty years. In some instances the progress of the disease is arrested and the patient may reach an advanced age.

(c) Mixed or Complete Leprosy.—The lesions peculiar to the tubercular and the anæsthetic forms develop simultaneously, or in succession. While the distinction between the two main forms is in well-marked cases sharply defined, there are many cases which must be referred to the mixed form.

Diagnosis.—The direct diagnosis of leprosy in the early stage may be difficult. The erythematous macules with hyperæsthesia, pain, and pigmentation, and the subsequent development of tuberculous nodules are characteristic. In the nervous form the areas of persistent anæsthesia, with bullæ, ulceration, deformities, and necrosis of the hands and feet, are important. A history of residence in a country in which leprosy prevails, even without actual association with known lepers, justifies the suspicion of contagion. The bacteriological examination of the nasal discharge, the serum of a blister, or of an excised nodule may settle a doubtful case.

The differential diagnosis involves the consideration of a great number of chronic affections which present resemblances to leprosy, among which may be named especially syphilis, lupus, multiple neuritis, syringomyelia, and Morvan's disease.

Prognosis.—The experience of history shows leprosy to be an incurable disease. Abortive cases occur but they are extremely rare. The prognosis as regards recovery or even as regards the arrest of the process is highly unfavorable; that as regards the expectancy of life must be guarded. The miserable life of the leper may be prolonged for twenty years or more. The outlook in the tuberculous form is less favorable than in the anæsthetic form.

XXXI. TUBERCULOSIS.

Definition.—An infectious disease caused by the Bacillus tuberculosis, and characterized histologically by the formation of tubercles and infiltrations of tuberculous tissue, which undergo caseation and necrosis or sclerotic changes; anatomically, by alteration and destruction of the parts immediately affected, and clinically, by local and constitutional symptoms which vary according to the structures involved and the extent of the process.

Etiology.—Predisposing Influences.—Tuberculosis is a wide-spread disease affecting both human beings and animals. Among the latter the domestic animals and, in particular, the bovines chiefly suffer. Wild animals in captivity are peculiarly liable to tuberculosis. The liability of the hog is much less than that of horned cattle; of the horse and sheep very slight; of the dog and cat even less, though these animals housed as pets with tuberculous persons sometimes contract the disease. Rabbits and guinea-pigs, especially the latter, are peculiarly susceptible to tuberculous infection, and are for that reason much used for the purposes of laboratory research. Avian tuberculosis constitutes a special variety of the disease. The wide *prevalence* of tuberculosis among human beings is due to methods of living favorable to the propagation of the infecting principle. It is most prevalent in the centres of population and in densely peopled localities in which direct sunlight, fresh air, and cleanliness are little known. About one-seventh of all deaths are caused by tuberculous disease. *Climate* has little influence as a predisposing cause. Tuberculosis is more common in proportion to the population in temperate than

in tropical or extreme northern regions. *Altitude* is important. The great plateaus of the United States and Mexico, the settlements of the high regions of the Alps, the Andes, and the Himalayas are remarkably free from tuberculous affections. *Soil and Locality.*—Tuberculous diseases and especially phthisis have been shown to be more prevalent in wet, badly drained districts than in dry uplands with a porous, sandy soil. The influence of soil in this respect consists in an increased liability to catarrhal affections and hence general increase in susceptibility. *Mode of Life.*—Tuberculosis is fostered by darkened houses and unventilated sleeping rooms. Habitual life in the open air is unfavorable to it alike in the individual and in the community. Like other readily transmissible infections, it spreads with great rapidity when introduced among aboriginal peoples previously free from it. *Race.*—The influence of racial susceptibility is much less than that exerted by the mode of life. It is difficult to separate these factors. The American Indian, the negro, and the immigrant peasant from Ireland and Southeastern Europe suffer in this country from an especial liability. There is an apparent relative immunity among the Jews. *Personal Predisposition.*—The phthinoid or pterygoid chest is not only seen in phthisical individuals but it is also characteristic of those who manifest a peculiar susceptibility to phthisis. On the other hand a very large proportion of those who contract the disease have well-developed chests and every evidence of perfect health. Now that tuberculosis is known to be an infection to which the liability is wide-spread, less attention than formerly is paid to the so-called diathetic states. *Age.*—Tuberculous infection may occur at any age. Early adult life, from twenty to thirty-five, is attended with a peculiar liability. The susceptibility of the various tissues and organs varies with different periods of life. In infancy the intestines, glands, and meninges, in childhood and adolescence the bones and lymph-nodes, and later the lungs, other viscera, and the skin are more commonly affected, but any form of tuberculosis may occur at any period of life. *Sex.*—Women become tuberculous in slightly higher proportion than men. *Occupation.*—Those who work in a confined and dusty atmosphere are very liable to contract pulmonary consumption. From this point of view phthisis merits a place among the occupation diseases. *Previous Disease.*—Catarrhal affections predispose to tuberculosis. It is to this fact that must be ascribed the frequency of pulmonary tuberculosis after the acute diseases in which bronchial catarrh plays a prominent part, as pertussis and measles. The marked predisposition to tuberculosis of the lymph-nodes on the part of young children is due to their liability to catarrhal processes in the upper respiratory passages and bronchi. I am not disposed to think that tuberculous infection is especially favored by enteric fever. Influenza and variola are regarded as predisposing influences. In such cases the development of the disease may be either the result of direct infection upon a soil prepared by the antecedent malady or of the lighting up of a latent tuberculous focus. Tabes mesenterica is doubtless in most cases the result of infection by way of the lesions of a catarrhal enterocolitis. Among chronic diseases syphilis and diabetes are very often followed by pulmonary tuberculosis, and the latter affection is a common terminal condition in chronic bronchitis, disease of the heart,

cirrhosis of the liver, and chronic nephritis. Cases of tabes and other forms of spinal sclerosis often terminate in pulmonary tuberculosis. A blow or injury is very often followed by tuberculous disease of the part. In meningitis, or bone or joint disease, it is necessary to assume an antecedent latent tuberculosis; when pleural or pulmonary tuberculosis follows a contusion of the chest in the absence of fracture of a rib or laceration of the pleura or lung, we may assume that the injury has rendered infection possible. Careful inquiry will often elicit a history of previous symptoms of tuberculosis. The danger of a surgical operation in local tuberculosis is always to be considered. Latent trouble elsewhere may be lighted up, or an acute miliary process develop.

The Pathogenic Organism.—The demonstration by Koch of the tubercle bacillus and the etiological unity of the tuberculous diseases constitutes one of the most remarkable and beneficent achievements of modern medicine and ranks in importance with Jenner's great work.

The bacilli are present in varying abundance in all the lesions of tuberculosis. They are very numerous in active lesions, but scanty in the sluggish processes of chronic glandular or bone disease. When a tuberculous focus, as a softened lymph-node, discharges its contents into a vein or lymph vessel, the bacilli are swept on with the current to effect new lodgment and produce new tubercles at various points in the body. When not found in the effusions of serous tuberculous inflammation, as pleurisy, or in sections of chronic or obsolescent lesions, the nature of the process may frequently be demonstrated by culture or inoculation.

There are four specific varieties of *Bacillus tuberculosis:* 1, the human; 2, the bovine; 3, the avian; and, 4, the bacillus of cold-blooded animals. These closely resemble each other in morphological characters. Tuberculous infection in man is almost always with the first variety, occasionally with the second, in rare instances with the third, and so far as is known not at all with the fourth. Koch (1901) stated that the bacillus of bovine tuberculosis does not cause the disease in human beings nor the bacillus of human tuberculosis the disease in cattle. Differences in the two forms are recognized; but the view that bovine tubercle bacilli may give rise to tuberculosis in man is now generally accepted. Individual strains differ greatly in virulence. The bacilli are thrown off by way of the discharges, the sputum in laryngeal and pulmonary tuberculosis, the fæces in intestinal tuberculosis, the urine, vaginal discharges and rarely the semen in the genito-urinary forms, and tuberculous sinuses, abscesses, and ulcerated surfaces in various parts of the body. They are present in scrofulous lymphnodes, the skin in lupus, and in the blood in generalized miliary tuberculosis. They retain their vitality outside the body for an undetermined period. The chief source of infection is the sputum. Beside this all others fall into insignificance. The principal vehicle of transmission as shown by Cornet is the atmospheric dust.

Mixed infections with other bacteria occur in cases attended with ulceration and especially in pulmonary tuberculosis. The common organisms are streptococci, pneumococci, and influenza bacilli. To these added or secondary infections much of the fever and progressive dyscrasia are to be ascribed.

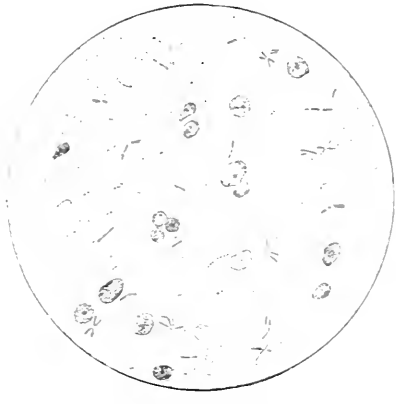


Fig. 273.—Spread of sputum showing Bacillus tuberculosis

Modes of Infection. — (a) *Heredity.* — Clinical and experimental evidence are alike against transmission from the male by means of the spermatozoids. The hypothesis that transmission may occur from the tuberculous mother by way of the ovum has some experimental support. Transmission by way of the blood, the bacilli penetrating the placenta, is supported by clinical and laboratory facts. The placenta under such circumstances is usually tuberculous, but in some instances it has been apparently normal. The number of reported cases of congenital tuberculosis in man is limited.

The difficulties in determining the part played by heredity in individual cases arise from the uncertainty in regard to the transmission of individual susceptibility from the affected forebears to the offspring, the absence as yet of definite conclusions as to the prolonged latency of tubercle bacilli in the tissues of an apparently healthy child of a tuberculous parent, and the fact that tuberculosis is at the outset a distinctly local process, which may, when acquired by postnatal infection, become circumscribed or obsolescent, and remain latent in the tissues (lymph-nodes, for example) for an indefinite period, to become again active under various circumstances, as traumatism, acute disease, or softening and rupture of a gland-capsule. The mere fact that a parent, grandparent, or collateral relation in a previous generation suffered from tuberculosis no more proves the hereditary nature of the disease than the occurrence of scarlet fever in them would render it hereditary. Yet the assumption in regard to the one disease might with the same indifference to scientific accuracy be made of the other.

(b) *Inoculation.*—Tuberculosis in man has been in rare instances produced by inoculation. Those who work in the post-mortem room frequently contract local skin tuberculosis, the nature of which has been demonstrated both microscopically and by inoculation in animals—post-mortem warts. These lesions are discrete, small nodules and are almost always situated upon the backs of the hands or fingers. Inoculation may also take place in various accidental ways; among these are circumcision, cuts from the broken spit-cup of a consumptive, and the bite of a tuberculous person. There is no evidence to support the assertion of the anti-vaccinationist that consumption is transmitted in the vaccine virus, beyond the fact that in a very limited number of cases lupus has developed at the site of the vaccine pock. In point of fact, the lesion almost always remains local at the seat of inoculation.

(c) *Inhalation.*—The vehicle is atmospheric dust to which bacilli are attached. This dust in rooms occupied by the consumptive and around his person is made up of the dried pulverized sputum. An obvious measure of prophylaxis is to keep the sputum, discharged into suitable receptacles, moist and disinfected until it can be destroyed by fire or otherwise effectually disposed of. In some instances direct infection takes place by the inhalation of tubercle bacilli contained in the fine particles of moist sputum ejected by the patient in the act of coughing or even in conversation, and remaining for a time suspended in the air in his immediate neighborhood. Among the facts that lend support to the hypothesis of infection by inhalation are the frequency with which the early lesions involve the larynx, lungs, and bronchial glands; the prevalence of the disease in cloisters,

asylums, prisons, and other similar institutions, and the remarkable investigations of Flick, who showed that not only certain localities but also particular houses become infected, and that members of different families successively occupying such houses succumb to the disease.

The degree of intimacy of association with the consumptive plays an important part in the danger of the transmission of the disease. It is no uncommon thing for a woman who has nursed a consumptive daughter or sister to contract the disease and die of it in the course of some months. This occurrence is infrequent among nurses, whose attendance is less close and hours are less prolonged. The latter have also an advantage in their technical knowledge of the dangers and in habits of prophylaxis. In the case of husband and wife this danger of the transmission of pulmonary tuberculosis is abundantly established by common observation and the results of statistical inquiries.

(d) *Infection by Food.*—The milk of tuberculous cows, and milk foods, including butter made from it, have been shown to be capable of giving rise to the disease. Tuberculous lesions of the udder are not necessary, the milk of animals healthy in this respect having been shown to be infectious. Bovine tuberculosis constitutes a positive danger to the human race. The frequency of tuberculosis of the intestines and mesenteric glands in young children finds a ready explanation in infected milk.

The meat of tuberculous animals is not without danger. It has been shown experimentally to be infective to guinea-pigs. Thorough cooking probably destroys the infecting principle; uncooked meats, smoked beef and similar articles may convey the disease. Legal enactments against the exposure for sale of the flesh of tuberculous animals are well founded.

A. Acute Miliary Tuberculosis.

Tubercle bacilli find their way into the blood from a focus of tuberculous endangitis or the perforation of the vessel wall by a softening caseous mass.

Varieties.—Three clinical forms occur: (a) the general or so-called typhoid, characterized by the symptoms of an acute general infection; (b) the pulmonary, in which the symptoms are chiefly referable to the lungs, and (c) tuberculous meningitis.

(a) **Generalized Miliary Tuberculosis.**—Miliary nodules are thickly disseminated throughout the various organs of the body.

Symptoms.—The symptoms are those of an acute general infection, and the condition presents many of the features of enteric fever, for which it is often mistaken. The signs of local disease are rarely marked. The onset is usually gradual; in some of the cases abrupt. Nose-bleeding is not common. The pulse is rapid, not often dicrotic, and shows remarkable variations in frequency within short periods of time. The fever increases, the temperature being very irregular, usually much higher in the evening than in the morning and often reaching 104° F. (40° C.) or more with remissions of three or four degrees. In some of the cases the range is intermittent. Inverse temperatures are observed. There is profound asthenia with rapid loss of flesh. Pulmonary symptoms, increased respiration frequency, especially early in the attack, and dyspnœa with faint

cyanosis, are common. Diffuse râles are heard, and in some of the cases there may be dulness at an apex, or patches of subcrepitant and crepitant râles at various parts of the chest. In a group of cases there is no fever or the temperature does not exceed subfebrile ranges, and the true nature of the disease is revealed only upon post-mortem examination. Delirium is less common than somnolence and stupor, which deepen to coma, terminating in death. Toward the last there may be an intensification of the lung symptoms, or the evidences of involvement of the meninges. Cheyne-Stokes respiration is common.

Diagnosis.—The DIRECT DIAGNOSIS of this form of acute miliary tuberculosis rests upon the signs of a profound toxæmia, irregularity of the temperature, the occurrence in some cases of an inverse range, the rapidity and irregularity of the pulse, and the absence of the signs which characterize the other specific infections and septic processes. If there be localized pulmonary signs, the history of glandular or bone tuberculosis, or meningeal symptoms, the diagnosis becomes more probable. Choroid tubercles, or tubercle bacilli in the fluid obtained by lumbar puncture, or in the blood, render it positive and final. Guinea-pig inoculations are uncertain and take time. Clough has recently grown tubercle bacilli in culture from the circulating blood of patients with miliary tuberculosis[1] (1917).

The DIFFERENTIAL DIAGNOSIS between general miliary tuberculosis and enteric fever often taxes the art of medicine to its utmost. This is the case when the onset is marked by bronchitis attended by dyspnœa and faint cyanosis, with little cough. Great rapidity and irregularity of respiration and pulse, and in general terms a correspondence in their frequency, irregularity of the temperature and conformity to the remittent or intermittent rather than the subcontinuous type, signs of deficient oxygenation of the blood, constipation, only slight enlargement of the spleen, herpes, the absence of rose spots, and a leucocytosis are in favor of miliary tuberculosis. The following facts must receive due consideration. There are cases common in childhood and not extremely rare in adult life in which the fever curve of enteric fever is remittent throughout. Constipation is by no means rare in enteric fever, and diarrhœa may occur in miliary tuberculosis. Enlargement of the spleen occurs in both diseases, but is neither so early nor, except occasionally in children, so decided in tuberculosis. Herpes, though rare, has been observed in enteric fever. Rose spots occur in tuberculosis, but they are rare and appear singly rather than in crops, and do not present the appearance nor run the course of the eruption of enteric fever. Leucocytosis is common in miliary tuberculosis; leucopenia the rule in enteric fever. But leucocytosis occurs in enteric fever complicated by inflammatory and suppurative processes. Albuminuria and the diazo reaction may occur in both diseases. The Widal reaction may not be conclusive, since it may occur in an individual who has passed through an attack of enteric fever and subsequently become tuberculous. Nor, for the same reason, is the presence of Bacillus typhosus in the urine conclusive. If that organism is, however, found in blood cultures the diagnosis of enteric fever may be made. The two conditions may coexist and the lesions of both diseases have been present upon post-mortem examination.

[1] Johns Hopkins Hosp. Bull., Vol. XXVIII, p. 370.

(b) **The Pulmonary Form.**—The acute phenomena develop in persons who suffer from persistent cough or are known to have chronic pulmonary tuberculosis, or, especially in children, may follow an infectious disease, as measles or whooping-cough.

Symptoms.—The onset is like that of an acute bronchitis or bronchopneumonia. Troublesome cough, mucopurulent expectoration, sometimes rusty sputum or blood spitting, dyspnœa, slight cyanosis, and a dusky flushing of the cheeks are among the symptoms that attract attention. In children especially, but sometimes also in adults, there are patchy areas of dulness at the bases posteriorly, with areas of tympanitic resonance, the sign of collateral emphysema. Râles of larger size, both sibilant and sonorous, may be heard; but much more commonly they are fine or coarse crepitant. A grazing friction scarcely to be distinguished from the finest subcrepitant or crepitant râles is the sign, as established by post-mortem observations, of a miliary tuberculosis of the pleura. Bronchial breathing of high pitch is, in children, often heard at the bases and opposite the root of the lung. The temperature is not usually very high, 102°–103° F. (38.9°–39.5° C.), often irregular, and may be of inverse type. The pulse is irregular. The spleen is usually enlarged. Toward the end cerebral symptoms sometimes develop, the pulse grows more feeble and rapid, coarse râles obscure the finer respiratory signs, and Cheyne-Stokes breathing occurs. The duration of the disease varies from a fortnight or more in the acute to several months in the chronic cases.

Diagnosis.—The DIRECT DIAGNOSIS is not usually attended with difficulty. In children this form of miliary tuberculosis very often follows measles and whooping-cough and constitutes the principal danger in those diseases. The history of chronic tuberculosis, pulmonary, glandular, or joint, is of diagnostic importance. Tubercle in the choroid, when found, establishes the diagnosis, but the proportion of cases in which it occurs is limited. The occurrence of meningeal symptoms is important. Tubercle bacilli are often absent from the sputum upon repeated examination; when found their significance is positive.

DIFFERENTIAL DIAGNOSIS.—This relates to non-tuberculous bronchopneumonia. The anamnesis is very important. A history of tuberculous disease in any of its forms, even a chronic cough without impairment of general health, merits careful consideration. The degree of dyspnœa and cyanosis, tubercle bacilli, hæmoptysis, and, above all, the persistence of the fever and other symptoms are of diagnostic value.

(c) **The Meningeal Form.**—*Acute Tuberculous Meningitis.*—An acute miliary tuberculosis in which the membranes of the brain, less commonly also those of the cord, are chiefly implicated. As the membranes of the base are the common seat of the process, the affection is sometimes spoken of as basilar meningitis. This form is especially common between the second and fifth years of life. It is rare during the first year and relatively infrequent in adult life. A primary tuberculous depot may usually be discovered, most frequently in the bronchial or mesenteric glands. It may be in the lungs, the middle ear, the bones, or genito-urinary organs. In a small proportion of the cases the most careful autopsy fails to reveal the local source of the infection.

Symptoms.—There are frequently prodromal symptoms. These consist of gradual loss of appetite and weight, irritability, fretfulness, and change of disposition. There may be a history of recent measles or whooping-cough, or of a fall.

Stage of Irritation.—The onset may be attended with a severe general convulsion, or with the usual triad of meningeal symptoms—headache, vomiting, and retraction of the muscles of the back of the neck, the last being less marked than in cerebrospinal fever. Fever is present, usually moderate but gradually increasing to 102°–103° F. (39°-39.5° C.). The pain is intense and paroxysmal, and the exacerbations are accompanied by a short sudden scream—*hydrocephalic cry*. The child holds its hand to its head, and sometimes screams continuously for hours at a time. The vomiting is without apparent cause and is repeated from time to time. The bowels are usually constipated. The retraction of the neck may be slight at first and only manifest when the head is bent forward or rotated. The respiration frequency remains normal, but the pulse, at first rapid, becomes irregular and slow. Sleep is restless and accompanied by muscular twitchings and sudden starts and cries. The pupils are as a rule contracted, and very often to a greater degree upon one side than upon the other. Kernig's sign is present.

Transitional Stage.—The signs of irritation gradually and irregularly subside. The vomiting ceases, the belly becomes retracted and scaphoid, and constipation is stubborn. Headache is replaced by dulness, stupor, and occasional delirium. The retraction of the neck continues, the pupils are dilated and irregular, and strabismus is common. There may be convulsions, or rigidity of various muscle groups. The respiration is at times sighing and irregular, and when disturbed the child utters the sudden, shrill cry so often heard in the disease. The temperature is irregular and atypical. Irregular patches of erythema are noted, and if the skin is tapped with the finger-tip, or the nail drawn across it, a vivid red spot or line shortly appears and only slowly fades—*tache cérébrale*—a sign of little diagnostic value since it occurs in enteric fever, hysteria, and other conditions in which there is relaxation of the peripheral vessels.

Stage of Paralysis.—The stupor deepens to coma and the patient cannot be aroused. Muscular spasms occur and there may be general convulsions. The pupils are dilated and irresponsive to light; there is paralysis of ocular muscles, and ophthalmoscopic examination reveals optic neuritis. Tubercles in the choroid are by no means always seen. Tetanoid contractions, cataleptic states, tremor, and athetoid movements occur, and in some cases there are hemiplegias or monoplegias. Aphasia may occur. The pulse now becomes rapid and feeble, and the symptom-complex known as the typhoid state, with dry tongue, muttering delirium, involuntary discharges, and subnormal temperature, develops. The duration of the attack varies from two to four or five weeks. There is sometimes a pre-agonistic rise of temperature.

A moderate leucocytosis is usually present during the whole course of the attack. There are cases which begin with great abruptness and intensity, in persons apparently in good health, and run their course in a few days; on the other hand there are cases which run a chronic course, with anomalous symptoms suggestive of tumor of the brain.

Diagnosis.—The DIRECT DIAGNOSIS of tuberculous meningitis rests upon the presence of the signs of a local tuberculous process, the mode of onset, which differs from that of cerebrospinal fever, the course of the disease, and the results of lumbar puncture. The fluid withdrawn is usually turbid and often contains tubercle bacilli. It is sometimes sterile.

DIFFERENTIAL DIAGNOSIS.—For the details of the differential diagnosis between tuberculous meningitis and pneumococcus and streptococcus meningitis and cerebrospinal fever see Cerebrospinal Fever.

B. Tuberculosis of the Lymph-nodes—Scrofula.

Tuberculosis of the lymph-nodes is more common in children than in adults, but it occasionally occurs in middle life and infrequently in persons of advanced age. Catarrhal inflammation of mucous membranes, by which their resistance is impaired, is probably the most important predisposing factor in this form of tuberculous disease. Tonsillitis and nasopharyngeal catarrh doubtless stand in a causal relation to the cervical adenitis so common in childhood; measles, pertussis, and recurrent attacks of catarrhal bronchitis, to tuberculosis of the bronchial glands; and the intestinal diseases to which infants are prone afford the gateway of infection of the mesenteric glands.

Glandular tuberculosis is very commonly a local form of the disease. There is a remarkable tendency to encapsulation and latency. The deposition of lime salts is common. A long quiescent bronchial gland may become the source of a local or general tuberculous process.

VARIETIES.—(a) Generalized, and (b) local tuberculosis of the lymph-nodes are to be considered.

(a) **The Generalized Form.**—A form of the disease in children characterized by the successive implication of groups of glands, and terminating in a general tuberculous cachexia or in meningitis has been described.

(b) **Local Tuberculous Adenitis.**—The groups usually affected are, in the order of their frequency, the cervical, the tracheobronchial, and the mesenteric. The cervical glands are very frequently involved. The children of the poor, and especially the negro, mostly suffer, but those living in affluence do not wholly escape. Chronic rhinitis, tonsillitis, otitis media, eczema capitis vel faciei, conjunctivitis, or keratitis may afford the port of entry for the infection.

Cervical Glands.— SYMPTOMS.—The submaxillary glands are most frequently and usually first involved. The posterior cervical chain, the glands above the clavicle, and the axillary glands are also affected in many cases. The disease may affect one or both sides; when both, to a much greater extent on one than the other. The bronchial glands may also be tuberculous. Infection of the pleuræ or lungs may subsequently take place. The enlarged glands may at first be felt as discrete, smooth, firm, and somewhat elastic tumors, over which the skin is freely movable. They rapidly enlarge and coalesce, forming large disfiguring masses to which the overlying skin becomes adherent, with subsequent inflammation and suppuration. If the resulting abscess be not opened, it breaks, leaving a sinus which heals slowly, followed by a characteristic, retracted,

unsightly scar. Fever is usually present during the active stage of the process and the patient is anæmic. The process is slow, but many of the cases, especially in children, ultimately recover.

Diagnosis.—*Direct.*—Indolent glandular enlargements in the neck and axillary region, more marked on one side than the other, becoming adherent and slowly softening with abscess formation, are usually tuberculous, especially in children who suffer from local catarrhal or inflammatory diseases of the upper air-passages, otitis media, eczema of the head or face, chronic conjunctivitis or keratitis, or tuberculous disease in other parts of the body.

Differential.—Slight cervical adenitis occurs in connection with various catarrhal processes and the exanthemata. As a rule these enlargements gradually undergo resolution as the primary disease subsides. Sometimes they suppurate. This shorter course of the process, the association with acute disease, and the slighter degree of enlargement are of diagnostic importance.

Fig. 274.—Chronic cervical adenitis.—Rotch.

Hodgkin's disease may at first be very difficult to recognize. The greater frequency of tuberculous adenitis in children, the early implication of the glands in the submaxillary region, the slow development, the tendency to inflammatory adhesions among the glands themselves and to the skin, and to suppuration and abscess formation, are in favor of the tuberculous nature of the process. Limitation to a group of glands, as the cervical or axillary, or to one side, is much more common in tuberculous adenitis than in Hodgkin's disease.

Tracheobronchial Glands.—This form of tuberculosis is very common in young children, and particularly so in the inmates of foundling asylums, orphanages, and similar institutions. The glands may attain large size. The trachea and bronchi may be flattened, and pressure may be exerted upon the superior cava, the pulmonary artery, and the azygos vein. The softening caseous contents of the glands may perforate into the bronchi or trachea and cause asphyxia; into the great vessels with general infection of the blood stream, or very rarely into the œsophagus. Pulmonary infection very often occurs either by contiguity of tissue or along the root of the lung. Pericardial tuberculosis may occur.

Symptoms.—Pressure symptoms occur, but they are less common and less urgent than the anatomical conditions suggest. The enlarged mass constitutes one of the forms of mediastinal tumor. Dyspnœa, paroxysmal, brassy cough from pressure on the recurrent laryngeal nerves, cyanosis and puffiness of the face from pressure on the superior cava, dysphagia from compression of the œsophagus, are occasional symptoms. In the majority of cases the mechanical disturbance is slight or absent altogether. Nor are definite physical signs common. Impaired resonance upon light percussion over

the manubrium sterni may be noted, and slight relative dulness along the spine in the upper dorsal region. These physical signs are, however, neither so constant nor so well marked as to serve a useful purpose in the diagnosis.

Mesenteric Glands—Tabes Mesenterica.—A slight enlargement is common and may not give rise to special symptoms. As a rule the enlargement is general and attains considerable size. The retroperitoneal glands are often coincidently involved. Caseation and softening occur. Resorption of the fluid portions and the deposition of lime salts sometimes take place. The tuberculosis may be primary, infection having arisen by way of the lesions of intestinal catarrh, or it may be secondary to tuberculous lesions of the bowel.

Symptoms.—Tabes mesenterica is common in very young children. The belly is enlarged and tympanitic; the enlarged glands cannot always be felt; there is diarrhœa with thin, watery, and offensive stools. The nutrition is deranged and the little patients are anæmic, puny, and wasted. The superficial abdominal veins are often enlarged and conspicuous. There is fever of hectic type. In a group of cases there is an associated tuberculous peritonitis, the belly is distended, firm, or doughy, and nodular tumors may be felt. Massive tuberculous enlargement of the mesenteric and retroperitoneal glands occasionally occurs in adults.

Diagnosis.—*Direct.*—In young children the diagnosis is commonly attended with no great difficulty. The appearance of the child is suggestive. Sometimes the enlarged glands are palpable. Tuberculous adenitis elsewhere, or the evidence of tuberculous disease of the lungs, is of diagnostic importance.

Differential.—The diagnosis of the essential character of circumscribed tuberculous glandular masses in the abdomen, especially when they are of considerable size, and in the adult without tuberculous disease of the intestines, peritoneum, or lungs, is often attended with difficulty. The differential diagnosis can in many of the cases only be reached by exclusion and may even then remain in doubt. Tuberculin may be used.

C. Tuberculosis of the Serous Membranes.

General Tuberculosis of Serous Membranes.—The process may be general, the pleuræ, pericardium, and peritoneum being involved simultaneously or in rapid succession. There may be an acute miliary tuberculosis; a more chronic form with agglomeration of tuberculous material, with caseation and inflammatory and suppurative lesions; and finally a chronic proliferative process with firm tuberculous nodules, fibroid lesions, great thickening of the membranes, and the absence of exudate. The pericardium is less frequently involved than the pleuræ and peritoneum.

Tuberculous Pleurisy.—The pleurisy may be acute, with fibrinous, serofibrinous, purulent, or hemorrhagic exudate; or it may be chronic. It is very often latent. Secondary and terminal forms occur. A rare form of acute tuberculous pleurisy with ulceration and necrosis of the pleura has been described. Subacute cases with a serofibrinous exudate are very common. They are almost constantly associated with circumscribed tuberculous disease of the lungs, or with tracheobronchial adenitis. The

exudate may become purulent. There are cases in which no signs of tuberculous disease can be found, in which after a period of latency varying from a few weeks to many years, with excellent health, pulmonary or acute miliary tuberculosis supervenes. The visceral pleura is always involved in pulmonary tuberculosis extending to the periphery of the lung. Adhesions with more or less thickening result. In the absence of protecting adhesions a caseating mass in the lung may perforate the visceral pleura and cause pyopneumothorax. Finally there is a chronic adhesive pleurisy with great thickening and involvement of the interlobar pleura and the lung itself. For the symptomatology and diagnosis of tuberculous pleurisy see Pleurisy.

Tuberculosis of the Pericardium.—The process may be part of a general miliary tuberculosis, or latent in cases of chronic tuberculosis or other chronic disease, or it may cause a chronic adhesive pericarditis analogous to the more common chronic adhesive pleurisy. There are acute cases with fibrinous or plastic, serofibrinous, hemorrhagic, or purulent exudate, and the ordinary symptoms of pericarditis, in which, in the absence of tuberculous disease elsewhere, the true nature of the process cannot be recognized *intra vitam*. (See Pericarditis.)

Fig. 275.—Tuberculous peritonitis. Outline indicates a hard mass which was found on operation to consist of areas of nodules matting together the intestines.—Rotch.

Tuberculosis of the Peritoneum. — There may be diffuse miliary tuberculosis or circumscribed areas corresponding to tuberculous ulceration of the intestine, acute miliary tuberculosis with serofibrinous or bloody exudate, chronic tuberculosis with agglomerations of tuberculous tissue undergoing caseation and necrosis, chronic proliferative or fibroid peritonitis with extensive adhesions and thickening of the capsule of the liver and spleen. The infection takes place by way of the intestines, especially in children, and in adults is propagated from the Fallopian tubes or the seminal vesicles, prostate, or testicle. In by far the largest proportion of cases infection of the peritoneum is secondary to tuberculosis of the lungs or pleura. Tuberculous peritonitis has been known to follow contusion of the abdomen; it sometimes has its starting point in the hernial sac and often constitutes a terminal condition in chronic visceral disease, especially cirrhosis of the liver.

Symptoms.—Tuberculosis of the peritoneum presents peculiar clinical phenomena which serve to distinguish it from peritonitis due to other causes.

The disease may be latent and discovered only upon operation, or post mortem. In other cases the onset may be sudden with urgent symptoms, as fever, vomiting, pain, and tenderness. There are cases in which the onset and early symptoms suggest enteric fever.

Fever occurs in the acute cases and the temperature often reaches 104° F. (40° C.) or more; in many of the cases the rise is only to subfebrile ranges—100° F. (37.8° C.). In the chronic cases fever is absent and subnormal temperatures often occur. The pulse is variable and, in the absence of fever, of moderate frequency. Tympanitis is common in the acute cases, but in the chronic form the belly may be small and doughy. Ascites of small amount is common; it is usually serous, but may be purulent or hemorrhagic, and is sacculated from the beginning, or soon becomes so. A diffuse pigmentation of the skin may suggest Addison's disease. Irregular attacks of pain associated with fever and digestive disturbance occur, and tenderness upon pressure is a more or less continuous symptom. Dense infiltration of the omentum with tubercles and fibrinous exudation may convert that structure into a thick, cord-like mass adherent to the transverse colon and extending across the abdomen. This tumor-like omental thickening may be recognized upon palpation. Similar masses may be felt in other parts of the abdomen. Sacculated fluid exudates confined by adhesions among the abdominal or pelvic organs, the mesentery, and the walls, form cyst-like tumors which suggest ovarian or other cysts and often lead to errors in diagnosis.

Less frequently the mesentery of the small intestine, the root of which extends in an oblique direction from the lumbar vertebræ to the right sacro-iliac symphysis, undergoes thickening and retraction, which is associated with great shortening of the intestine and thickening of its walls in such a manner that the bowel is drawn together into a tumor-like mass occupying the right side of the abdomen. These changes may be so extensive as to involve the bowel in its entire length. Massive enlargement of the mesenteric glands is very often present in tuberculous peritonitis, especially in children.

Diagnosis.—The DIRECT DIAGNOSIS of tuberculosis of the peritoneum is attended with much difficulty in the acute cases with sudden onset, great pain and tenderness, rigidity of the abdominal muscles, vomiting, and fever. The presence of tuberculous lesions elsewhere, and in particular at the apex of one lung, in the pleuræ, bones, or genito-urinary tract, is of great diagnostic importance. In this group of cases prompt operative measures are indicated, not only in order to clear up the uncertainty as to the causal conditions—*surgical diagnosis*—but also as the only curative measure which yields promise of relief in several of the conditions, as perforation of the bowel or other hollow viscus, or the rupture of an abscess or cyst, which cause acute peritonitis with precisely the same symptoms. In the subacute forms the direct diagnosis cannot always be made. Persistent abdominal symptoms in a tuberculous individual constitute sufficient ground for a provisional diagnosis, which the subsequent course of the case will frequently confirm. Many of the subacute cases are latent.

The diagnosis in the chronic forms depends upon the presence of the general symptoms of chronic peritonitis, the recognition of tuberculous foci in other parts of the body, encysted fluid exudate, or irregular tumor-like masses within the abdomen, the tuberculin test, which may be used in any doubtful case unattended by fever or with fever of only moderate range, and the finding of tubercle bacilli in the fluid obtained by paracentesis, or a positive reaction to the injection of such fluid into guinea-pigs.

Differential.—The points of discrimination between peritonitis due to other causes and tuberculous peritonitis have been indicated in the foregoing paragraphs. Non-tuberculous neoplasms are usually more local, circumscribed, and definite in their relation to the viscera, as the kidneys, spleen, or liver. Fever is less apt to occur. But more important still are the absence of prior or concurrent evidences of tuberculosis elsewhere, and negative results of laboratory investigations.

The omental and intestinal tumors which occur in the chronic forms are fairly distinctive and only lead to uncertainty in the case of malignant disease. The differential diagnosis rests upon the anamnesis, the more rapid wasting in cancer, and the differences in the cachexia of the two conditions.

Ovarian Cysts.—Errors of diagnosis are common. In some of the cases of tuberculous peritonitis with encysted exudate the general health is fairly well preserved. The physical signs may be similar in both conditions. The contour in tuberculous pseudocysts is less regular; areas of dulness upon percussion, or palpable nodular masses may be demonstrable,

Fig. 276.—Tympany due to tuberculous peritonitis.—German Hospital.

and changes in form or position may arise with variations in the amount of gas in the coils of intestines. Depression of the vault of the vagina occurs in both conditions. Tubal disease and nodular masses in one or both ovarian regions are suggestive. Febrile outbreaks are common in tuberculosis, but rarely occur in non-inflammatory ovarian cysts.

Cirrhosis of the Liver.—If the ascites is so great as to interfere with the palpation of the liver in a doubtful case, paracentesis is necessary for diagnostic purposes. A hemorrhagic fluid may be present in tuberculosis or carcinoma. This occurrence, together with thickening of the peritoneum or demonstrable tumors, or the evidence of tuberculosis or carcinoma in distant organs is diagnostic. In a considerable proportion of the cases of hepatic cirrhosis, tuberculosis of the peritoneum occurs as a terminal condition.

D. Tuberculosis of the Alimentary Canal.

Tuberculous lesions of the structures forming the digestive tract, with the exception of the liver and intestines, are rare.

Lips, Tongue, and Mouth.—Tuberculous ulcers of these organs occur in rare instances, mostly in association with laryngeal or pulmonary disease. Upon the lips they are liable to be mistaken for chancre or epithelioma. Tuberculous ulcers upon the tongue occur in the form of deep circumscribed

lesions, with well-defined but irregular borders and a caseous base. They resist treatment, not being influenced by the iodides, and tend to spread. The glands at the angle of the jaws are not enlarged. The salivary glands are very rarely the seat of tuberculous infection. Tuberculosis of the hard and soft palate in rare instances is the result of the invasion of these structures from adjacent parts. The tonsils are frequently infected. There may be superficial ulceration or diffuse infiltration with miliary tubercle. Caseous depots may be present. Infection may take place by means of tuberculous milk or other food, dust, or by the sputum in pulmonary disease. The frequency of tuberculous cervical adenitis, especially in children, finds an explanation in tonsillar disease. In ulcers of doubtful character upon the lips and tongue, or elsewhere in the mouth, a portion of the tissue may be excised for examination, or inoculations may be made. In a suspicious ulcer of the tongue failure of the iodides and absence of glandular involvement are against a diagnosis of syphilis. A Wassermann test should be made.

Pharynx and Œsophagus.— In laryngeal and chronic pulmonary tuberculosis miliary tubercles and superficial ulceration frequently invade the oropharynx. The latter condition, when associated with ulceration of the epiglottis, is attended with great pain upon deglutition, and constitutes a most distressing condition in laryngeal phthisis. Adenoid vegetations of the nasopharynx are in some instances infected. An extension from the larynx may invade the upper part of the œsophagus.

Stomach and Intestines.—Ulceration of the wall of the stomach is a recognized pathological condition but the diagnosis is not often made during life, since the symptoms are the same as in ordinary peptic ulcer. It has occasionally been observed post mortem in tuberculous subjects, but non-tuberculous peptic ulcer is more liable to occur in those debilitated and rendered anæmic by tuberculous disease. The probability that a peptic ulcer may become tuberculous is to be considered. Intestinal tuberculosis may be primary, especially in children, and is then usually followed by infection of the mesenteric glands or peritoneum. Primary tuberculosis of the intestine in the adult is exceedingly rare.

Symptoms.—Irregular diarrhœa, colicky pains, and moderate fever occur. Intestinal hemorrhage may be the initial symptom. Emaciation and signs of tuberculosis of the lungs or elsewhere suggest the actual pathological condition. There are cases in which the tuberculosis begins in the cæcal region, and the symptoms are circumscribed tenderness, slight irregular fever, and diarrhœa alternating with constipation. When these symptoms subside and recur after quiet intervals of varying duration, the condition simulates a chronic appendicitis. Hemorrhage occurs and necrosis may take place, causing peri-appendicular abscess or perforation into the peritoneum. Thickening of the intestinal wall forms part of the process.

Secondary lesions are much more common. The lower portion of the ileum and the large bowel are usually involved. Infection occurs by means of the swallowed sputum, and the intestinal disease gives rise to troublesome and distressing symptoms in the later stages of many cases of phthisis. The lymphatic glands are early involved and there is frequently extensive ulceration of the mucous membrane of the small and large bowel. There

may be ovoid ulcers in the ileum, corresponding to Peyer's patches, but as a rule the tuberculous ulcer is transverse and in many cases annular. Its borders and floor are thickened from the infiltration of tubercle, which shows caseation at various points. The muscular coat is often involved, patches of recent tubercles are seen upon the corresponding serosa, local adhesions occur, forming knot-like masses among the intestinal coils, and in rare instances perforation takes place. Sclerotic changes often proceed side by side with caseation and necrosis, and lead to cicatrization, irregular puckering, and stenosis. These lesions are sometimes localized in the cæcum and appendix, and form dense sausage-shaped tumors in the right lower quadrant of the abdomen, slightly or not at all movable, painful upon palpation, and suggestive of carcinoma. More extensive adhesions and infiltration in this region sometimes occur, and in rare instances a fecal fistula. *Fistula in Ano*—*Anal Fistula.*—This condition is in a large proportion of the cases tuberculous and associated with pulmonary tuberculosis which is sometimes latent or obsolescent. Operation is occasionally followed by a flaring of the lung trouble into activity, whether *post hoc* or *propter hoc* cannot always be determined. This fact does not militate against effectual operation by excision, since it is better to suffer from one focus of tuberculosis than from two.

Secondary tuberculous ulceration of the intestine manifests itself by a group of abdominal symptoms superadded to those of the pre-existing disease, usually pulmonary. Less frequently the intestinal lesions are secondary to tuberculosis of the peritoneum, the primary infection being in the lymph-nodes in children, or the genito-urinary tract in adults of either sex. Abdominal pain, tenderness, loss of elasticity with local doughiness or tumor formation, particularly in the right iliac region, diarrhœa often alternating with constipation, and later the signs of stenosis of the bowel, namely, local bloating, smooth sausage-shaped tumors indicating the contour of the distended gut, and stormy peristalsis, make up the clinical picture. In the rare cases in which the obstruction becomes complete the ominous characteristic symptoms of occlusion of the bowel (q.v.) appear.

Diagnosis.—The DIRECT DIAGNOSIS of primary tuberculosis of the intestine cannot always be made even in children. It depends upon hereditary predisposition, the possibility of feeding upon the milk of tuberculous cows, irregular high fever, rapid emaciation and loss of strength, and the presence in the stools of many tubercle bacilli upon repeated examination. Secondary lesions may be diagnosticated when persistent abdominal symptoms, not yielding to treatment, come on in the course of pulmonary consumption or local tuberculosis in other parts of the body, and in particular when there are also localized physical signs indicative of intestinal thickening, kinking, or obstruction. If fecal fistula develops and tubercle bacilli are found in the discharge as well as in the stools, the diagnosis is positive.

DIFFERENTIAL. — The discrimination between intestinal tuberculosis and the conditions which resemble it cannot in all cases be made. Two topics, however, demand especial mention—carcinoma and appendicitis.

Cachexia and pain occur as in carcinoma elsewhere. Fever is not a prominent symptom. The temperature is on the contrary often subnormal.

Ribbon-shaped stools, foul-smelling stools in which blood, pus, and necrotic fragments of the neoplasm are found, and the general symptoms of stenosis are suggestive of cancer. Absence of tuberculosis elsewhere, negative findings as to bacilli, and failure of the temperature rise after the injection of tuberculin are of great diagnostic importance.

There are rare cases of tuberculosis of the cæcum in which the process invades the lymphoid tissue of the appendix—tuberculous appendicitis. Primary attacks of appendicitis are so well characterized that the question of tuberculosis does not enter into their consideration. The acute or subacute character of the early symptoms even in the chronic cases would appear in the anamnesis. Of diagnostic importance are other diffuse abdominal symptoms, pain, tenderness, diarrhœa preceding the local phenomena, and the coincidence of tuberculosis in other organs. It is not to be forgotten that an attack of non-specific appendicitis may develop in a tuberculous individual.

E. Tuberculosis of the Brain and Spinal Cord.

Tuberculosis occurs as an acute meningitis which, while chiefly basilar, is almost always also spinal, and constitutes one of the manifestations of the acute form of general or disseminated infection—acute miliary tuberculosis (q.v.); as a chronic meningo-encephalitis due to the development of multiple tubercles, usually within circumscribed limits; and finally as solitary tubercles (see p. 686).

F. Tuberculosis of the Genito-urinary Organs.

Tuberculosis of the genito-urinary tract is frequent and important. Lesions have been observed in the fœtus, and the occurrence of tuberculous orchitis in very young children suggests the possibility of hereditary transmission. In the preponderating majority of instances the disease is secondary to disease of some distant organ, especially the lungs, and the infection must be ascribed to transmission by way of the blood. In a considerable proportion infection takes place from the peritoneum. Tubal and vesical tuberculosis have, however, been observed in cases of intestinal tuberculosis in which no evidence of the implication of the peritoneum could be found. Less frequently the disease arises by direct infection from the rectum to the bladder, or to the uterus or vagina, in consequence of adhesions and fistula formation. Tuberculous abscesses in the pelvis may be the source of infection of any of the genito-urinary organs. Vertebral tuberculosis may implicate the kidney by direct extension. The possibility of primary tuberculosis as the result of direct infection in sexual intercourse appears very great. Whether or not accidental infection by way of the vagina or urethra may take place from other sources, as infected instruments or syringes, suppositories, or in digital examination, or by transmission from the rectum by way of the clothing has not been fully established. The infection may involve any of the tissues of the genito-urinary system. It often extends rapidly, and in some cases there are manifestations of the disease at different points at the same time.

Tuberculosis of the Kidneys.—The disease may be secondary. In acute general tuberculosis scattered tubercles are present in the substance and upon the surface of the kidneys. In pulmonary tuberculosis there may be scattered nodules, or pyelitis. Primary tuberculosis of the kidney also occurs. In many of the cases the lesions are at the same time present in the kidneys, extending to the pelvis and uterus, and in the bladder, prostate, and seminal vesicles, and the seat of primary invasion is uncertain. Renal tuberculosis is most frequent in middle life but may be met with at any age. Males suffer much more frequently than females.

Symptoms.—The urine contains pus in varying amounts. There is increased frequency of micturition. These symptoms often go on for years without abnormal subjective sensations and with maintenance of the general health. There may be tenderness upon firm pressure. In exceptional cases the kidney may be greatly enlarged, or there may be a pyonephrosis. Under such circumstances there may be a palpable abdominal tumor. The urine is albuminous, and in addition to pus-cells contains epithelium and granular débris. Tube-casts are not very common. Tubercle bacilli are present. Hemorrhage may occur. As the disease advances the other kidney becomes involved, and a tuberculous cachexia with chills, irregular fever, sweating, and emaciation and progressive asthenia ensues. The lungs are implicated and an acute disseminated miliary tuberculosis occurs as a terminal event. Encysted caseous or calcareous masses in the kidney are occasionally found in the post-mortem room and point to the possibility of spontaneous cure.

Diagnosis.—Direct.—The above symptoms, associated with the evidence of tuberculosis in the testicle or prostate, or in the tubes or ovaries, the presence of tubercle bacilli in the urine, and a positive reaction to the tuberculin test, justify a positive diagnosis. The differentiation of the urine by catheterization of the ureters renders possible a diagnosis of the kidney affected. The urine may contain bacilli from tuberculous lesions in the bladder or elsewhere in the genito-urinary tract, and the fact that the morphological and tinctorial characters of the smegma bacillus are practically the same as those of the tubercle bacillus is to be borne in mind. The specimen for examination in a doubtful case must be obtained by catheterization under the strictest precautions against contamination, and the possibility that even then smegma bacilli may be accidentally present must not be forgotten. Inoculation tuberculosis caused by the urinary sediment is proof positive of genito-urinary tuberculosis, but not necessarily of tuberculosis of the kidney.

Differential.—It may be difficult to differentiate tuberculous pyelonephritis from calculous pyelitis. A history of attacks of renal colic, various forms of crystalline sediment and blood-cells in the urine, or actual hemorrhage, are in favor of the latter. Hemorrhage is much less common in tuberculosis of the kidneys.

Suprarenal Capsules.—Tuberculosis of the adrenals with fibrocaseous lesions is the most common anatomical change found in Addison's disease, and may manifest itself by the symptoms of that disease (q.v.).

Tuberculosis of the Ureters and Bladder.—The symptoms of renal tuberculosis are those of cystitis, and infection of the bladder is usually secondary to infection of the kidneys on the one hand, or of the testes,

prostate, or seminal vesicles on the other. The process very often invades the ureters from the pelvis of the kidney. Primary tuberculosis of the bladder is a rare affection.

Tuberculosis of the Prostate and Seminal Vesicles.—These organs are frequently the seat of tuberculous growths and caseous nodules. The prostate is often found upon digital examination to be enlarged and nodular. It is sometimes tender. There is great irritability of the bladder, vesical tenesmus, frequent micturition or retention of urine, in which case the use of the catheter is attended with great pain. Tuberculosis of the urethra is rare. It may present the symptoms of stricture.

Tuberculosis of the Testes.—The diagnosis is usually unattended with difficulty because the organ is accessible and the changes are somewhat characteristic. The disease occurs in infants as well as in adults. One or both testicles may be involved. It may be primary, but in most cases is secondary to pulmonary or other visceral or bone tuberculosis. It is frequently associated with tuberculous peritonitis. The tuberculous testicle may be recognized by the enlargement which principally affects the epididymis, pain, tenderness, and only a moderately uneven surface.

The DIFFERENTIAL DIAGNOSIS between tuberculous and syphilitic disease of the testicle may be attended with uncertainty. In the latter, pain and tenderness may be absent, the testicle itself rather small, the epididymis involved, and the surface, owing to the agglomeration and various size of the gummata, is more nodular and uneven.

Tuberculosis of the Fallopian Tubes and Ovaries.—The tubes are very frequently affected. The disease is often primary. There is enlargement with great thickening and infiltration of the walls, upon which, in some cases, irregularities of the surface may be felt. It may occur in children and young girls, and is usually bilateral. The ovaries are secondarily involved. Abscess formation and the extension to the peritoneum are common. Implication of the uterus is extremely rare.

Diagnosis.—DIRECT.—This rests upon the local findings, such as enlargement and irregular thickening of the tubes, evidences of adhesions, signs of peritoneal tuberculosis or pulmonary phthisis, anæmia, loss of weight, fever in the evening upon moderate exertion and at the menstrual period.

DIFFERENTIAL. — Gonorrhœal salpingitis may be present without serious derangement of the general health. The enlargement of the tubes is not attended with the same degree of infiltration or irregularity of the surface, the anamnesis is suggestive, and the presence of gonococci in the discharges conclusive.

Tuberculosis of the Liver, Spleen, Myocardium, Endocardium, and Arteries cannot be recognized with certainty during life. These forms of visceral tuberculosis are therefore rather of anatomical than of clinical interest.

G. Tuberculosis of the Lungs.

Pulmonary Tuberculosis; Phthisis; Consumption.

VARIETIES.—(a) Acute pneumonic phthisis; (b) chronic ulcerative phthisis, and (c) fibroid phthisis.

(a) ACUTE PNEUMONIC PHTHISIS.

According to the distribution of the lesions two types are recognized, the pneumonic and the bronchopneumonic.

The Pneumonic Form. — A single lobe or an entire lung may be involved. This form is much more common in adults than children, and in males than females.

Symptoms.—The onset is usually abrupt, with a chill followed by high fever, pain in the side, cough, and expectoration, at first scanty and mucoid, later more abundant, often frothy and blood-stained. The attack frequently occurs in the midst of apparent health; occasionally during the course of an apparently ordinary mild influenza or "cold," and sometimes in an individual who has a tuberculous lesion regarded as obsolescent. The respiration is rapid and dyspnœa may be urgent; the pulse is frequent and variable. The physical signs are those of croupous pneumonia, feeble vesicular murmur, with crepitus, later dulness, increased vocal fremitus, and bronchial breathing. They correspond to the limits of a lobe or to a whole lung, and when, as is often the case, they are also, in the course of some days, found upon the opposite side the clinical picture is that of a double pneumonia.

Diagnosis.—The DIRECT DIAGNOSIS rests upon the course of the attack and the finding of tubercle bacilli in the sputa. The latter have been observed as early as the fourth day. As a rule their presence is not noted until later. Fibres of elastic tissue constitute the signs of necrosis of the pulmonary structure. The following clinical manifestations are suggestive and should arouse a suspicion as to the character of the process: hereditary predisposition to tuberculous infection; individual history of tuberculous infection which may have remained subacute or become quiescent; physical depression preceding the outbreak, especially if accompanied by cough and expectoration; an irregular temperature range conforming rather to the remittent than the continuous type; recurrent chills; circumscribed patches of high-pitched, ringing, coarse crepitant râles persisting for several days with but little change; a mucopurulent greenish expectoration, and extremely feeble breath sounds over the affected region.

The DIFFERENTIAL DIAGNOSIS between acute pneumonic tuberculosis and croupous pneumonia cannot be made in a large proportion of the cases during the first week. There is, in truth, usually no suspicion that the case is not one of ordinary pneumonia until even a longer time has elapsed.

Prognosis.—The outlook is in the highest degree unfavorable. Death has occurred as early as the sixth day, more commonly after three or four weeks, or as late as the second or third month. In a limited number of cases the acute symptoms gradually subside and the case becomes one of chronic phthisis.

The Bronchopneumonic Form.—The lesions are those of an acute caseous bronchopneumonia. Groups of lobules are affected, with crepitant tissue intervening, but extensive areas or even an entire lobe may be involved.

A second form is due to the aspiration of blood and the contents of tuberculous cavities into the finer bronchial tubes during hæmoptysis —tuberculous aspiration pneumonia. This condition may follow early

hæmoptysis, which has not been preceded by marked symptoms, or occur after hæmoptysis in the course of a chronic tuberculosis.

In a third form the caseous bronchopneumonia involves lobules at both apices and in other parts of the lungs, causing patches of consolidation varying in diameter from 1 to 3 or 4 cm. and sometimes scattered uniformly throughout both lungs.

Symptoms.—These are the forms which constitute a majority of the cases of acute pulmonary tuberculosis—*phthisis florida* or *galloping consumption.* They are common in adults but far more common in children. The clinical picture varies greatly. In adults the disease may develop in persons apparently well or in those who have been failing in weight and strength. The onset is rapid but not abrupt. There are irregular chills, fever of hectic type, sweating, loss of appetite, cough, and expectoration which is usually slight. Blood spitting is sometimes the first event to attract attention. The pulse and respiration frequency are high, and loss of weight and strength is rapid and progressive. The physical signs may be at first obscure, but presently diminished expansion, patchy dulness, especially at an apex, vesiculobronchial respiration, and moist crepitant and small mucous râles occur. The disease affects one, more commonly both lungs. Tubercle bacilli and fibres of elastic tissue are present in the sputa, often at an early date. The symptoms become more intense, and in the course of two or three weeks in the more acute cases the patient falls into the so-called typhoid state, with stupor, delirium, dry tongue, and high fever. Meanwhile the physical signs, more extensive dulness, bronchial breathing, high-pitched and coarser râles, indicate the extension and progress of the lesions. In some cases, however, the signs are obscured by the development of areas of collateral emphysema. Softening may occur with cavity formation and corresponding changes in the physical signs. In children this form of pulmonary tuberculosis may arise as an independent disease. Much more commonly it follows an acute infection, especially measles and pertussis. In a majority of the cases the bronchopneumonia which occurs as a sequel to diseases of this group is tuberculous.

Diagnosis.—The DIRECT DIAGNOSIS of acute bronchopneumonic phthisis rests upon the association of the foregoing symptoms and physical signs with the presence of elastic fibres and tubercle bacilli in the sputa.

The DIFFERENTIAL DIAGNOSIS between tuberculous and non-tuberculous bronchopneumonia is, in the early stages of the disease, and especially in children, often impossible. Later the presence of elastic-tissue elements and tubercle bacilli are decisive. Meanwhile the anamnesis is important. A hereditary predisposition, association with individuals who are tuberculous, or dwelling in an infected house, or enlarged superficial lymphatics, or a history of symptoms or signs indicative of enlarged bronchial glands is important. The signs of marked apical lesions are highly suggestive of tuberculous disease, but diffuse tuberculous bronchopneumonia may occur without marked apex consolidation.

Prognosis.—The outlook is in the highest degree unfavorable. In adults death may occur in the course of three or four weeks; in children within a few days. There are cases, however, in which the disease runs a somewhat more protracted course, and a limited number, both in adults and children, in

which after the gravest symptoms the condition of the patient undergoes some improvement and the case gradually passes into one of chronic phthisis.

(b) CHRONIC ULCERATIVE PHTHISIS.

This is the common form of chronic pulmonary tuberculosis.

The lesions vary greatly in kind, distribution, and extent. They comprise nodular and miliary tubercles, tuberculous bronchopneumonia, pneumonic inflammation of the vesicular structure surrounding the tubercles, frequently presenting the appearance of ordinary red hepatization, sometimes the more uniform diffuse tuberculous infiltration, cavities of various size, together with collateral emphysema and changes in the bronchi and bronchial glands and in the pleura, with firm, thick adhesions or effusion, which may be serofibrinous, purulent, or hemorrhagic. The tendency is, (a) to caseation, softening, ulceration, necrosis, and cavity formation, and (b) to sclerosis. The latter process may result in the formation of a limiting membrane, by which the lesion is encapsulated, or, when extensive, in traction deformities of the chest, and bronchiectasis.

The fact that the primary lesion or lesions in pulmonary tuberculosis of the chronic ulcerative type are local and circumscribed is of the greatest practical importance both in diagnosis and prognosis; first, because it underlies the clinical division of the cases into incipient and advanced, and second, because cases referable to the first group are mostly amenable to treatment.

The distribution of the lesions is in a majority of the cases as follows: The earliest lesions are situated, not at the extreme apex of the lung, but 2 to 4 cm. below it and nearer the posterior and lateral surfaces than the anterior surfaces. Extension from this point is downward and forward, the upper lobe being progressively involved in regions corresponding to the first, second, and third interspaces, and spreading upon both sides of the midclavicular line. Less commonly the primary lesion is found in the upper lobe at a point corresponding to the first and second interspaces below the outer third of the clavicle. As the process extends downward, the anterolateral region of the lobe is involved. Invasion of the middle lobe of the right lung is usually by extension from the upper lobe. Secondary implication of the lower lobe begins at a point 2 to 4 cm. below its apex at the level of the fifth dorsal spine, and extends downward and outward in a line roughly corresponding to the inner border of the scapula when the patient's hand is placed upon the opposite shoulder and the elbow raised as high as possible. In the course of time the upper lobe of the opposite lung usually becomes involved, the earliest lesions appearing a short distance below the actual apex and rapidly becoming diffused. The right upper lobe is first involved somewhat more commonly than the left. Primary implication of the base is rare.

In advanced cases miliary tuberculosis, visceral tuberculosis involving various organs, amyloid disease, and fatty liver occur.

The extension of the lesions is, (a) peripherally by the direct invasion of contiguous tissue; (b) radially by means of the lymph current; (c) by conveyance along the bronchial system, (i) in the direction of adjacent or distant vesicular structures—inhalation, insufflation; (ii) in the direction

of the upper air-passages—laryngeal ulceration; (d) by transference, *e.g.*, to the digestive tract—secondary pharyngeal, lingual, or intestinal tuberculosis; by dissemination, as in the case of the rupture of a gland or other encapsulated focus into a serous cavity, or a blood-vessel—acute miliary tuberculosis.

The progress of the lesions is variable. On the one hand, infiltration, caseation, softening, ulceration, necrosis, proceeding at different rates in different foci; while, on the other hand, sclerotic changes encapsulate and limit the advance of the disease and tend to circumscribe the process.

Symptoms.—As the primary infection is local, there is usually a period of latency. *The patient is tuberculous before he is consumptive, and, in the fortunate cases, he may be tuberculous without ever becoming consumptive.*

The MODE OF ONSET is determined by the degree of activity of the tuberculous process and the nature of the reaction of the infected individual. It may be characterized by latency, with indefinite symptoms not suggestive of pulmonary disease, or masked by the symptoms of grave disease in other organs, tuberculous or non-tuberculous.

Gastro-intestinal Symptoms.—Loss of appetite, gastric irritability and vomiting, acid eructations frequently precede the pulmonary symptoms for a considerable time. The cough is regarded as "a stomach cough."

Anæmia.—In children and adolescents, especially young girls, there is early chloro-anæmia with pallor, progressive weakness, palpitation and headache upon exertion, and slight afternoon fever. Menstrual irregularities, especially amenorrhœa, are suggestive.

Ague-like Fever.—Constitutional symptoms, recurring chills, fever, and sweating characterize the onset in a considerable group of cases. When such paroxysms recur with regularity and with only slight cough and expectoration, especially in a malarious region or in an individual who has previously suffered from ague, a false diagnosis may readily be made.

Pleurisy.—The early phenomena may be those of a persistent dry pleurisy, the signs of which are sometimes restricted to the apex, sometimes more extended. In other cases the impairment of health begins with pleural effusion. The resorption or aspiration of the fluid is sooner or later followed by the signs of consolidation in an upper lobe, and the symptoms of phthisis. Many of these cases are, in fact, pleurogenous, with secondary pulmonary infection, and the early dry cough is that of pleural irritation. In some cases the lung lesions rapidly develop; in others an interval of weeks, months, or even years may occur.

Hæmoptysis.—Blood spitting may be the first indication of the disease. An abundant hemorrhage is sometimes followed by the rapid development of the signs of a diffuse tuberculosis. In other cases hæmoptysis recurs from time to time before the positive physical signs of pulmonary disease can be recognized. It is probable that the local lesions almost always antedate the pulmonary hemorrhage.

Bronchitis.—The great majority of cases begin with the signs of a catarrhal bronchitis. The patients often suffer from nasopharyngeal catarrh and manifest an especial tendency to "catch cold." At length the cough becomes persistent, there is habitual expectoration, especially in the morning, and upon examination the râles, which are heard widely

over the chest, are found to be more abundant and moist and of higher pitch over the upper part of one lung, where there is also relative dulness and deficient expansion.

Chronic Bronchitis and Emphysema.—The terminal tuberculosis so common in these conditions is usually masked for a considerable time by the symptoms and signs of the primary condition. This is especially the case when there are asthmatic symptoms.

Laryngitis.—The symptoms of pulmonary phthisis are frequently preceded by hoarseness, occasional aphonia, and a laryngeal cough. It is probable that, in the majority of these cases, tuberculous lesions already exist in the lungs.

Tuberculosis of the cervical and axillary lymph-glands may precede the development of pulmonary tuberculosis for a long time, or coexist with quiescent lesions in the lungs.

Stages.—The attempt to divide the course of the attack into a stage of the growth and development of tubercles, a stage of caseation and softening, and a stage of cavity formation has fortunately been abandoned. In the first place, as new foci of disease are constantly forming in advancing cases, all three of these anatomical conditions are frequently present at the same time; secondly, they do not correspond with definite clinical periods, and, finally, a patient in the so-called third stage, with signs of cavity formation, is often in a more favorable condition, with better prospect for the arrest of his disease, than another in the first stage, with extensive and rapidly advancing infiltration or diffuse foci.

The following schema was adopted by the National Association for the Study and Prevention of Tuberculosis in 1905:

Incipient (favorable)	Slight initial lesion in the form of infiltration limited to the apex or a small part of one lobe. No tuberculous complications. Slight or no constitutional symptoms (particularly including gastric or intestinal disturbance or rapid loss of weight). Slight or no elevation of temperature or acceleration of pulse at any time during the twenty-four hours, especially after rest. Expectoration usually small in amount or absent. Tubercle bacilli may be present or absent.
Moderately advanced	No marked impairment of function either local or constitutional. Localized consolidation moderate in extent with little or no evidence of destruction of tissue; Or disseminated fibroid deposits. No serious complications.
Far advanced	Marked impairment of function, local and constitutional. Localized consolidation intense; Or disseminated areas of softening; Or serious complications.
Acute miliary tuberculosis	
Unimproved	All essential symptoms and signs unabated or increased.
Improved	Constitutional symptoms lessened or entirely absent; physical signs improved or unchanged; cough and expectoration with bacilli usually present.
Arrested	Absence of all constitutional symptoms: expectoration and bacilli may or may not be present; physical signs stationary or retrogressive; the foregoing conditions to have existed for at least two months.
Apparently cured	All constitutional symptoms and expectoration with bacilli absent for a period of three months; the physical signs to be those of a healed lesion.
Cured	All constitutional symptoms and expectoration with bacilli absent for a period of two years under ordinary conditions of life.

Trudeau's classification is as follows:

1. *Incipient.*—Cases in which both the physical and rational signs point to but slight local and constitutional involvement.
2. *Advanced.*—Cases in which the localized disease process is either extensive or in an advanced stage, or where, with a comparatively slight amount of pulmonary involvement, the rational signs point to grave constitutional impairment or to some complication.
3. *Far Advanced.*—Cases in which both the rational and physical signs warrant the term.
4. *Apparently Cured.*—Cases in which the rational signs of phthisis and the bacilli in the expectoration have been absent for at least three months or who have no expectoration at all; any abnormal physical signs remaining being interpreted as indicative of a healed lesion.
5. *Arrested.*—Cases in which cough, expectoration, and bacilli are still present, but in which all constitutional disturbance has disappeared for several months; the physical signs being interpreted as indicative of a retrogressive or arrested process.

"Closed" and "Open" Pulmonary Tuberculosis.—Too much stress has been laid upon the importance of tubercle bacilli in the sputa in the early diagnosis. These organisms do not appear until after the caseation and softening of a tuberculous lesion situated near a bronchus or bronchiolus, into which tuberculous material finds its way by the necrosis of the intervening tissue. The period prior to this event, which may extend over weeks or months, or, in extreme cases, over years, is known as the "closed period"; that which follows as the "open period." The general recognition of this distinction is desirable.

Symptoms of Incipient Pulmonary Tuberculosis.—The greater number of these have already been described under the heading "mode of onset." The association of hæmic, circulatory, digestive, and nervous derangements is especially important.

The *pulse* is either frequent but regular, or subject to abnormal acceleration upon physical effort or mental excitement. The *temperature* shows slight elevation upon exertion, after meals, and before and during menstruation. Observations for diagnosis must be taken every two hours during the day, while the patient is in repose. Subfebrile ranges—99.5° F.—are significant. *Chest pains* are common. They are of two kinds, pleural pain over the seat of a lesion, and a dull shoulder pain extending down the arm and sometimes mistaken for rheumatism. The *cough* is frequently short and dry, a troublesome hacking brought on by exertion or excitement or changes of external temperature. Very often it occurs only on rising in the morning and persists in paroxysms until a small, tough mucoid mass is expectorated, after which it is absent for the rest of the day. *The Sputum.*—There is little characteristic in the expectorated material. It is usually at this stage of the disease merely a grayish sago-like mucus, containing alveolar cells which have undergone the myelin degeneration. In the closed stage tubercle bacilli are absent, though one or two may, in rare instances, be found as the result of inhalation. Repeated examinations are necessary. Their continuous presence in the sputum is the positive sign of tuberculosis in the open stage. The examination may yield negative results for long periods in cases of quiescent limited upper lobe lesions, and then, after an attack of some acute affection, as influenza, or, in the midst of apparent health, bacilli may appear suddenly and last a short time—transient open tuberculosis. Elastic fibres are not often encountered in the incipient stages.

Hæmoptysis.—This accident occurs in about 70 per cent. of all cases of phthisis at some period in the course of the disease. The hæmoptysis which occurs in the incipient stage differs from that in the advanced stages in being, as a rule, slight, recurrent, and due to oozing from patches of acute congestion surrounding closed tuberculous foci, or to superficial erosions of bronchial mucosa. In advanced phthisis the bleeding is due to the erosion of a vessel in the wall of a cavity, or the rupture of an aneurism of a branch of the pulmonary artery. It is usually profuse and not rarely fatal. The expectorated blood in early hæmoptysis—closed tuberculosis—does not usually contain tubercle bacilli; that in the advanced stages is often followed by expectoration containing those organisms. Large initial blood spittings may, in rare instances, usher in the open stage and be associated with bacillary sputum.

General Nutrition and Weight.—The toxins which give rise to anæmia, vasomotor derangements, pseudodyspepsia, fever, and nervous erethism interfere with nutritive processes and cause loss of weight which is often rapid and striking.

Associated Diseases.—The patients are especially prone to catarrhal and other inflammatory outbreaks. Coryza, laryngitis, bronchitis, pneumonia, and pleurisy are common and may recur repeatedly in the same patient.

Hoarseness, due to subacute laryngeal catarrh with slight abductor paresis, may be an early symptom. Actual paralysis of the recurrent is less common. It may be due to pleural adhesions or to pressure upon the recurrent by tuberculous lymph-glands. It much more frequently occurs upon the left side. Phenomena of inferior diagnostic importance are unequal dilatation of the pupils, a reddish or bluish gingival line, slight or transient enlargement of the thyroid gland, and albuminuria.

The Physical Signs in Incipient Pulmonary Tuberculosis. — *Inspection* reveals very early a retarded and slightly diminished respiratory excursus in the infraclavicular region of the affected side. This sign may, in some cases, be earlier detected by palpation. The vocal fremitus may also be slightly increased. *Percussion* may show quite early relative dulness, often slight but recognizable by the higher pitch and shorter duration of the sound, and a slightly tympanitic quality. *Auscultation* yields even more suggestive signs. There is an early deviation from the normal type of breathing. The first change consists in the development of the quality described as rough. The inspiratory murmur is enfeebled. Cog-wheel or interrupted breathing is occasionally heard in the region immediately adjacent to and below the portion of the lung involved. This may, however, occur in other conditions. Moist crepitant and small mucous râles are early signs, but in many cases they remain long absent. Râles, not heard upon full inspiration, even full inspiration after coughing, may in some cases be elicited by a full inspiration followed by forced expiration, with cough at the end of the latter. The rough breathing is presently replaced by vesiculobronchial respiration which, as the lesion progresses, becomes bronchovesicular and, later, as consolidation becomes complete—advanced stage—bronchial. Pleural friction sounds of varying quality and intensity may often be heard over the affected region. They are sometimes transient, sometimes persistent. Basal friction sounds are also occa-

sionally heard in the incipient stage. Less frequent but very suggestive when present, is a subclavian systolic murmur, more common, as a rule, upon inspiration, though occasionally heard with expiration. This loud systolic whiff, due to traction upon the vessel wall by pleural adhesions, is a very striking phenomenon. Extension of the absolute cardiac dulness to the right or left, as the case may be, constitutes an important sign.

The Diagnosis of Incipient Pulmonary Tuberculosis. — Direct Diagnosis. —In the absence of cough and expectoration a positive diagnosis can rarely be made. When these phenomena are present, and especially when tubercle bacilli are found, the question as to the nature of the process is at once settled. From the standpoint of therapeutics the recognition of phthisis in the closed stage is of such importance that, in a suspected case, a provisional diagnosis constitutes a motive for immediate and systematic treatment. This provisional diagnosis rests not upon any single rational symptom or physical sign of the stage of incipiency, but upon the association of several of them in an individual in whom no other pathological process by which to explain them can be demonstrated. The anamnesis is important. Family predisposition, close habitual association with tuberculous persons, an unfavorable occupation, an unhygienic life may appear as etiological factors, but their absence has only a negative value.

The bodily conformation may be misleading. The classical *habitus phthisicus*—The phthinoid or paralytic chest—is usually symptomatic of advanced, not of incipient phthisis. It may be in some instances constitutional and indicate a predisposition to phthisis rather than actual tuberculous infection. Recurrent hoarseness, bronchitis, anæmia, dyspepsia, loss of weight, fever, and hæmoptysis are symptoms of great moment. Among the physical signs, diminished and retarded respiratory excursus of that part of the chest corresponding to the limited lesions, diminished resonance with a faint tympanitic quality, rough or vesiculo-bronchial respiration, and a few moist, clicking râles or a prolonged whizzing râle at the end of the first two or three inspirations are highly significant. It is to be remembered that in slowly advancing lesions the signs may be obscured by collateral emphysema. Not less significant is the localization of those signs in an infraclavicular or axillary region. The variation of auscultatory signs must be remembered—the fact that râles are sometimes heard only after cough, the frequent temporary disappearance of crepitus after several deep inspirations, the tendency of râles to disappear late in the day and in dry weather, and the intensification of auscultatory phenomena during menstruation. In any case in which the symptoms and signs warrant a provisional diagnosis, the patient must be carefully instructed as to his mode of life, the necessity of keeping himself under systematic observation, and the importance of the repetition of the examination at stated intervals.

Differential Diagnosis.—The mode of onset is to be considered. The patient is in failing health; has he the symptoms and signs of phthisis, latent or marked? Are his symptoms those which the toxins of tuberculosis cause? Superficial examination and hasty observation will not discover the answer to these questions. Above all, the practitioner must avoid the delusion that every paroxysmal fever is malarial and every cough in a dyspeptic a "stomach cough." *Pleurisy* is highly suspicious. Prob-

ably two-thirds of the cases of persistent fibrinous pleurisy or pleural effusion ultimately become tuberculous. *Malaria* may be recognized by the blood examination. *Hæmoptysis* is common in mitral disease, especially stenosis, and many cases of valvular disease of the heart are diagnosticated phthisis. This error may be avoided by a routine physical examination, which must in every case include the heart. *Cervico-axillary adenitis* may long coexist with a fair degree of health. It is well, however, to watch the

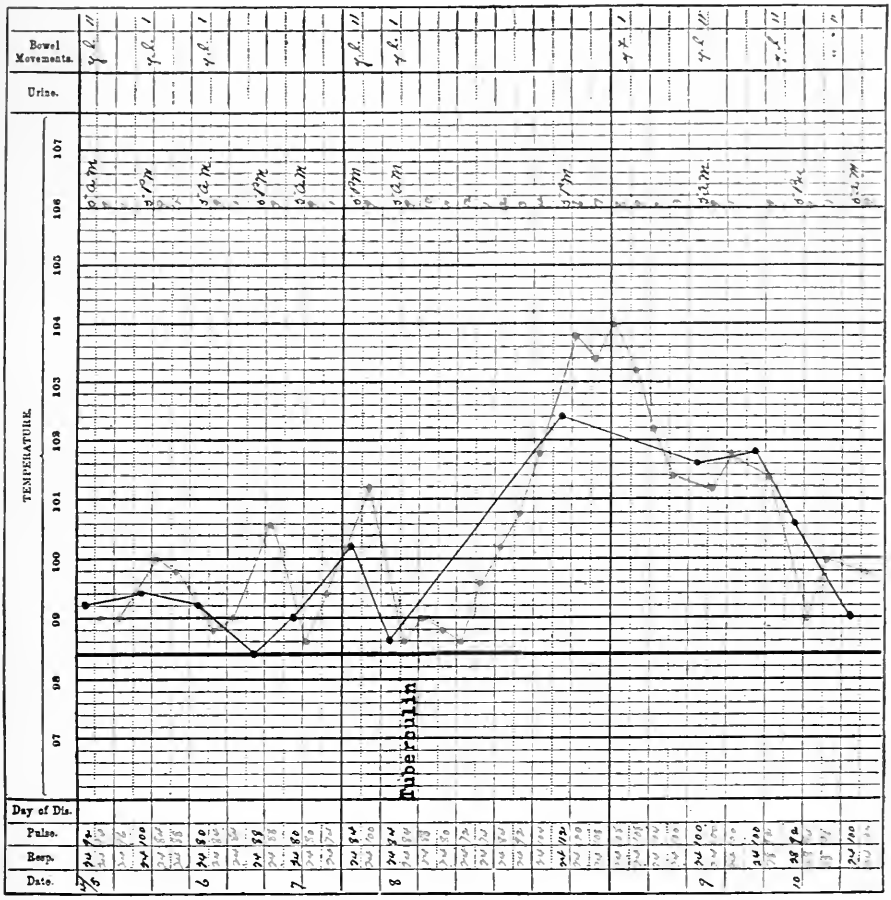


FIG. 277.—Subcutaneous tuberculin test. Positive reaction.

infraclavicular regions, especially upon the side in which the tuberculous glands are situated. *Laryngitis.*—Persistent hoarseness with swelling of the arytenoids and slight adductor paresis is frequently the phenomenon of pulmonary phthisis. *Recurrent catarrhal and inflammatory affections* in the respiratory tract are ominous, especially when there are hereditary or personal predisposing factors to tuberculous infection. Infraclavicular or axillary localizations are highly suggestive. The patient should be forewarned and placed under treatment. Such reassuring phrases as "local bronchitis" and "spot on the lung" are worse than obsolete; they are dishonest.

The Tuberculin Test.—If a case remains doubtful, or if, for personal reasons, delay must be avoided, tuberculin may be employed. To the objection as to danger, it may be affirmed that in the hands of innumerable careful observers its cautious use has not been followed by untoward effects. To the objection as to results, it is to be admitted that it has sometimes failed in cases of demonstrated tuberculosis, and that some degree of reaction has occurred in non-tuberculous cases. No one, however, has reported an intense reaction to tuberculin in a healthy person or in other diseases. The mode of procedure is as follows: The patient is kept in bed and the temperature taken every two hours from 8 A.M. to 10 P.M. for two days. At the end of this period and in the early morning, at first .5, then 2 and finally, if required, 5 mg. of Koch's old tuberculin are injected at intervals of three days. In children the dose is .10 to .5 mg., according to the age. The temperatures are taken at intervals of two hours as before. The constitutional reaction shows itself in fever which rises rapidly—102° to 104° F. (39°–40° C.)—and only gradually subsides. So long as it continues the patient must be kept in bed. Local reaction is frequently manifest in the suspected chest area in the form of fine moist râles. As this may occur in the absence of the febrile reaction, auscultation must be performed twice daily. Sputum previously absent may be ejected during or after the reaction and may contain bacilli. In other cases intense general and local reaction may occur without expectoration.

The Ophthalmotuberculin Test.—The method proposed by Calmette and Wolff-Eisner consists in the instillation of a drop of a 1 per cent. tuberculin solution into the eye, which is followed by a conjunctival hyperæmia in infected individuals, while in non-tuberculous individuals no signs of irritation follow.

Fig. 278.—Eye-dropper with throttle for measured drop.—Baldwin.

In Baldwin's clinical studies a filtered sterile normal saline solution of two strengths and a measured drop (0.025 c.c.) were employed, the latter to insure accuracy of dosage. A throttled eye-dropper with a calibrated mark to 0.025 c.c. was used, the throttle permitting the control of the liquid when filled to the mark. The technic is as follows:

"The two solutions of 0.33 per cent. and 0.5 per cent., respectively, were employed successively in each eye. When the first failed to react the stronger was instilled into the other eye after forty-eight hours. By this method severe reactions may be avoided in cases of suspected tuberculosis, though if time failed the stronger solution might be used without serious discomfort being anticipated should a marked reaction follow. In order to avoid any danger of contamination the solutions were sealed in glass tubes containing three or four drops and then boiled. These can easily be broken in a piece of gauze or cotton at a file mark. The eye-dropper is then inserted, after being cleansed with alcohol and sterile saline solution. Care is used to prevent the introduction of spicules of glass. The solution should be warmed in cold weather.

"The lid of one eye is pulled down and the measured drop instilled as with any other fluid by holding the eye-dropper parallel to the eye, but care should be observed that it does not overflow on the cheek; this is easily accomplished by holding the lid down until the drop is distributed about the sac."

BALDWIN'S SCHEME FOR RECORDING REACTIONS.

Negative.—No difference in color when lower eyelids are pulled down and compared.

Doubtful. Slight difference with redness of caruncle.

Positive. + = Distinct palpebral redness with secretion.

" ++ = Ocular and palpebral redness with secretion well marked.

" +++ = Deep injection of entire conjunctiva with œdema of lids, photophobia, and secretion.

TABLE SHOWING RESULT IN 137 CASES—Baldwin.

I. Pulmonary tuberculosis. No. cases, 45.		Reacted....42 Doubtful... 1 Negative... 2	(1 miliary and 1 tuberculin-treated).
II. Pulmonary or other tuberculosis; healed from 1 to 17 years. No. cases, 9.		Reacted.... 8 Doubtful... 1	(17 years).
III. Pulmonary or other tuberculosis suspected. No. cases, 26.	(a) From history (14 cases).....	Reacted.... 4 Doubtful... 4 Negative... 6	
	(b) From symptoms (8 cases).....	Reacted.... 2 Negative... 6	
	(c) From physical signs (4 cases)....	Reacted.... 2 Negative... 2	
IV. Apparently healthy persons. No. cases, 57.	(d) With family history of tuberculosis (18 cases).	Reacted.... 8 Doubtful... 1 Negative... 9	
	(e) Constantly associated with tuberculous doctors, nurses, husbands and wives of invalids (18 cases).	Reacted.... 6 Negative...12	
	(f) With no family history or exposure (21 cases).	Reacted.... 2 Negative...19	

Contraindications.—These include diseases of the conjunctiva, eyelids, and cornea, and of the internal structures of the eye. Undue exposure to dust, smoke, or strong light should be avoided during the test. The test is superfluous when the diagnosis can be made by physical or microscopic examination. The repetition of the test is not unattended with danger due to sensitization.

CUTANEOUS TUBERCULIN REACTION.—Von Pirquet, who suggested this test, employed at first a 25 per cent. solution of old tuberculin, but subsequently used the undiluted old tuberculin. The inoculation is performed on the forearm with a chisel-shaped instrument, the skin having been previously thoroughly cleansed with ether. Any suitable instrument may, however, be employed, and any convenient part of the body selected.

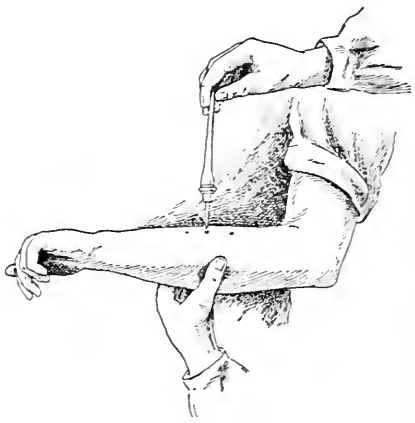
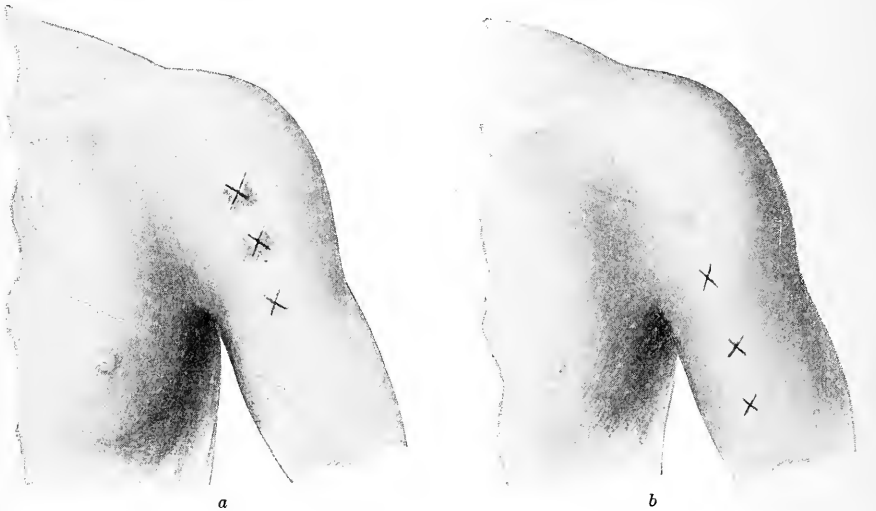


FIG. 279.—V Pirquet's method.

Positive Reaction.—If a papule of at least 5 millimetres in diameter develops at the point of vaccination in twenty-four hours, the patient may

be regarded as infected with tuberculosis. As a general rule the more active the tuberculous process the more intense and extensive the cutaneous reaction. Very intense reactions occur in glandular tuberculosis and in these cases inflammatory changes in the skin beyond the borders of the papule often occur—scrofulous reaction. The signs of reaction occur in periods varying from two or three hours to several days and are later in proportion to the degree of dilution of the tuberculin. No direct information as to the period at which the infection has taken place, namely, as to whether it is old or recent, its situation in the body or the extent of the lesions, is afforded by the cutaneous test.

Negative Reaction.—Failure may be due to absence of tuberculous infection or to various causes, among which von Pirquet especially enumerates the following: (1) relative insusceptibility, which is rare in early



a *b*

FIG. 280.—Petruschky's method: *a*, lower vaccination 1:10 negative, upper two 1:0 positive; *b*, lower vaccination 1:10 and upper two 1:0 negative.

childhood but not infrequent in older persons having circumscribed lesions which are encapsulated; (2) failure of the capacity for reaction in the last stages of tuberculosis; (3) loss of the capacity to react in immunity produced by tuberculization, either by progressively increasing doses or by a recent single dose of larger amount; (4) temporary disappearance of the capacity of reaction during an attack of measles.

In a considerable proportion of cases the failure of the test is, therefore, due to relative insusceptibility, and it has been shown that well-marked "secondary positive reaction" may be obtained upon the repetition of the test in these cases. A primary reaction is, however, the sign of an active tuberculous process. Petruschky makes, on the upper arm with the point of a cannula, cross-lined vaccinations with old tuberculin in dilutions of 1 to 10, 1 to 5, or 1 to 0. He finds no reaction in fully cured cases, and that the test is without danger and does not give rise to any important symptoms in any case. Petruschky regards this procedure as an important means of early diagnosis.

Tuberculin Inunction—Moro's Test.—This diagnostic procedure, described as "a diagnostic measure without rupture of continuity of the skin," consists in the inunction into the skin of the chest or abdomen, over an area of 4 square inches, of a mass about the size of a pea of an ointment composed of 5 c.c. of Koch's old tuberculin rubbed up with 5 grammes of anhydrous wool fat. Absorption takes place slowly, and on the following day, or, more commonly, not until the second day, in cases of previous or present tuberculous infection, a positive reaction shows itself in the appearance of small papules in the area of inunction or its immediate vicinity. These lesions vary in number and color from a few pale papules to a numerous, thick-set crop that are very red. The skin may be reddened and the seat of some itching. These cutaneous lesions wholly disappear in the course of a week. They are not attended by other local or constitutional phenomena. The test is negative when the skin shows no changes of any kind.

The Opsonic Method.—A. E. Wright and S. T. Reid,[1] in a communication "On the Possibility of Determining the Presence or Absence of Tuberculous Infection by the Examination of a Patient's Blood and Tissue Fluids," have arrived at the following conclusions:

"(1) Conclusions which can be arrived at when we have at disposal the results of a series of measurements: (a) Where a series of measurements of the opsonic power of the blood reveals a persistently low opsonic power with respect to the tubercle bacillus, it may be inferred, in the case where there is evidence of a localized bacterial infection which suggests tuberculosis, that the infection in question is tubercular in character. (b) Where repeated examination reveals a persistently normal opsonic power with respect to the tubercle bacillus, the diagnosis of tubercle may, with probability, be excluded."

"(2) Conclusions which may be arrived at where we have at disposal the result of an isolated blood examination: (a) Where an isolated blood examination reveals that the tuberculo-opsonic power of the blood is low, we may—according as we have evidence of a localized bacterial infection or of constitutional disturbance—infer with probability that we are dealing with tuberculosis—in the former case with a localized tubercular infection, in the latter with an active systemic infection. (b) Where an isolated blood examination reveals that the tuberculo-opsonic power of the blood is high, we may infer that we have to deal with a systemic tuberculous infection which is active, or has recently been active. (c) Where the tuberculo-opsonic power is found normal, or nearly normal, while there are symptoms which suggest tuberculosis, we are not warranted, apart from the further test described below, in arriving at a positive or a negative diagnosis."

The further criterion to which reference was made in the preceding paragraph is the following: When a serum, after it has been heated to 60° C. for ten minutes, is found to retain, in any considerable measure, its power of inciting phagocytosis, we may conclude that "incitor elements" have been elaborated in the organism, either in response to autoinoculations occurring spontaneously in the course of tubercular infection, or, as the case may be, under the artificial stimulus supplied by the inoculation of tubercle vaccine.

[1] Proceedings of the Royal Society, B Vol. LXXVII, 1906.

Iodine Test.—The iodine salts, and especially potassium iodide in moderate doses, may produce the physical signs of a local catarrh over a suspected area (Striker). The signs of consolidation may also become more definite. In the expectoration, which almost always follows, tubercle bacilli are frequently found.

X-ray Examination.—The results obtained are less definite than in the advanced stages of the disease. The complicated apparatus and great technical skill required also stand in the way of the general employment of this method in the diagnosis of pulmonary tuberculosis in the incipient stage.

The pulmonary tuberculosis which so often occurs as a terminal condition in pneumonoconiosis, chronic bronchitis, and emphysema is not usually recognizable in the incipient stage—masked tuberculosis. The symptoms of the primary disease are not so much altered as intensified. As the process advances tubercle bacilli previously absent may be found in the sputa.

Symptoms of Moderately and Far Advanced Pulmonary Tuberculosis.—There is no definite border-line between the stages. The difficulties in diagnosis disappear. The symptoms and signs become definite and characteristic. Any tyro can interpret the clinical picture.

1. Pulmonary Symptoms.—*Cough* may be slight but is usually prominent and annoying. After cavity formation it is commonly paroxysmal; with laryngeal involvement, husky and brassy. *Sputum.*—The expectoration is variable. There may be little or none, even when cough, fever, and rapid wasting are associated with the physical signs of extensive consolidation. The mucoid expectoration of the early period presently shows scattered grayish or grayish-green purulent masses in which tubercle bacilli and elastic fibres may be found. With softening the expectoration becomes more profuse and distinctly purulent. Nummular sputa are often present after cavities have formed. The sputa sometimes contain calcareous masses varying in diameter from 1 mm. to 1 or 2 cm., often of irregular shape. They are the result of the deposition of lime salts in circumscribed caseous masses. They find their way into a bronchus by the ulceration and necrosis of the intervening tissue. *Hæmoptysis.*—The amount varies from a trace to 500–750 c.c. In a majority of the attacks it does not exceed 15 c.c. It is in many cases repeated, and a hemorrhagic form of phthisis has been described. Hemorrhage into a large cavity may prove fatal without any blood being expectorated. Bacilli and elastic fibres may sometimes be discovered in the clots. After a day or two small black clots and blood-casts of the smaller bronchi are often coughed up and an access of fever may occur. *Dyspnœa.*—The respiration is not usually increased except upon exertion.

2. Constitutional Symptoms.—*Fever* is an important initial symptom. It may be remittent in type, or intermittent and paroxysmal, with ague-like periodicity. The temperature is an important indication of the progress of the disease. The periods of quiescence are afebrile and marked by gain in weight, while those of activity are accompanied by fever and loss of flesh. There are, however, rare cases in which, with advancing lesions, pyrexia is absent. The fever of the incipient stage is sometimes continuous, with slight daily remissions and exacerbations; that of the

moderately advanced stage corresponds to the activity of the process, and when present is of remittent or intermittent type, tending to subside altogether when the patient is kept at rest; while that of the far advanced period of the disease, with ulceration, necrosis, and the formation of cavities, is septic in character,—so-called hectic,—the range of temperature being subnormal—95°–96° F. (35°–35.5° C.)—in the morning between 10 A.M. and noon, and steadily rising to a maximum of 104°–105° F. (40°–40.5° C.) between 6 and 11 P.M. Colliquative sweating frequently attends the morning fall of temperature. The measurements should be made at two-hourly periods in order to ascertain the actual minima and maxima. Inverse temperatures are sometimes observed. *Sweating.*—Profuse sweats may occur, not only toward morning, but at any time at which the patient sleeps. They may occur early but are much more common in the far advanced cases. *Circulation.*—The pulse-frequency is increased and variable. It usually rises with the fever. The pulse is often large, soft, and compressible. As the sign of vasomotor paresis, capillary and venous pulsation may sometimes be seen. *Loss of weight* is a marked symptom. It is often rapid and extreme—*consumption; phthisis.* During periods of quiescence it is often arrested, and in favorable cases weight is regained. In rare cases increase of weight occurs in the absence of other signs of improvement. *Anorexia, vomiting, intractable diarrhœa, œdema* of the legs and feet with or without albuminuria, are common in the stadium ultimum. *Peripheral neuritis,* showing itself in extensor palsy of the wrists, more commonly the feet, sometimes occurs. The mental condition in the terminal dyscrasia is often remarkable—*spes phthisica.* The patients up to the very last busy themselves with plans for the future, new methods of treatment, different climates, business schemes, and the confident expectation of recovery.

Physical Signs in Advanced Pulmonary Tuberculosis.—*Inspection.*—The thorax undergoes deformities corresponding to the progressive diminution in the volume of the lungs. It tends to assume permanently the expiratory form.

Palpation.—Diminished expansion at the apex may be determined in the following manner: The examiner stands behind the patient, who is seated, and gently grasps the shoulders with his hands, the tips of the fingers being in the infraclavicular spaces, the thumbs resting upon the upper part of the scapulæ. The patient then slowly draws a deep breath; to study the expansion at the bases the hands grasp the two sides of the chest in a similar manner in the lower axillary regions. Lagging and limitation of the excursus are signs of great significance. The vocal fremitus is increased over areas of consolidation; over vomicæ it may be increased or diminished. It is usually enfeebled over thickened pleuræ.

Percussion.—In doubtful cases percussion should be performed during quiet breathing and upon full-held inspiration, as minor differences in the sounds upon the two sides then become more marked. Slight relative dulness may be recognized by the elevation of pitch which accompanies it. Light percussion above, beneath, and over the clavicle should be practised. The supraspinous fossæ and the points corresponding to the apices of the lower lobes are important regions. Dulness over consolidation and tympany over vomicæ is the rule; but scattered small tuberculous foci with

intervening air-containing vesicular structure, especially when there is collateral emphysema, yields resonance with a tympanitic quality, and a cavity filled with fluid will often yield a dull or, upon very nice percussion, a flat percussion sign. Over large cavities, situated near the periphery of the lung, the cracked-pot sound may be obtained.

Auscultation.—In the incipient stage the vesicular murmur is usually enfeebled, sometimes scarcely audible. Rough breathing is an early sign. Cog-wheel inspiration is often present, but it occurs in other conditions. With advancing lesions vesiculobronchial, bronchovesicular, and bronchial respiration succeed each other, to be finally replaced, as softening occurs and vomicæ form, by cavernous or amphoric breathing. Râles of all kinds, from the crepitus of the beginning lesion to the gurgling of cavities, attend the process. Râles are due largely to the accompanying bronchitis, and vary in kind and number according to the character of the secretion and the activity of the process in different parts of the lung. Puerile respiration may be heard over the adjacent unaffected lobes or over the opposite lung. The vocal resonance is increased and bronchophony and pectoriloquy may be elicited over areas of dense consolidation and cavities. Whispering pectoriloquy is an important sign of large superficial cavities. Pleural friction sounds are an important early sign and occur from time to time during the progress of the case. At first usually near the apex, they occur over advancing lesions in all points of the chest. Pleural friction at the left anterior border of the lung, and especially over the lingula, frequently has the cardiac rhythm—pleuropericardial friction. The signs of cavities are very variable. Situated superficially, a cavity may cause a distinct shallow depression in one or two intercostal spaces. A rapidly formed cavity, or several small cavities without much surrounding condensation or pleural thickening, may yield a full, clear resonance in which the only modification is a slightly tympanitic quality—vesiculotympanitic resonance. Tympanitic and amphoric resonance are usual. The pitch may be modified by opening and closing the mouth—Wintrich's sign—or upon change of posture—Gerhardt's sign. The cracked-pot sound can be brought out only over fairly large cavities with thin walls and superficially situated, and disappears for a time after it has once or twice been heard. Cavernous respiration is usually soft and low pitched. It may, however, be tubular or amphoric. Gurgling râles are common over large cavities and sometimes have a ringing or metallic quality, especially during coughing. Increased vocal resonance and whispering pectoriloquy are valuable signs. Over large cavities in the upper lobes the heart sounds and sometimes a transmitted systolic murmur are occasionally heard, and in rare cases sharp, splashing râles having the cardiac rhythm. Over a layer of dense, consolidated lung extending to a large bronchus there may be signs—circumscribed tympanitic percussion note, amphoric respiration, and coarse moist râles—which closely simulate those of a cavity. Light percussion, the pitch of the sound not being raised by opening the mouth or change of posture, and the absence of the cracked-pot sound may be of use in the recognition of the actual condition.

Diagnosis of Advanced Pulmonary Tuberculosis.—The DIRECT DIAGNOSIS rests upon the physical signs and the presence of tubercle bacilli in

the sputa. The symptoms are variable and acquire diagnostic value only in proportion as they correspond with the signs of the lesions and their distribution and evolution.

The spirometer has deservedly fallen into disuse in the diagnosis of incipient tuberculosis of the lungs, and its use is attended with danger in the advanced cases.

Röntgenography and Röntgenoscopy.—X-ray examination, though of small value in the detection of the early lesions of incipient pulmonary tuberculosis, is an important aid in the recognition of the position of the pathological changes and their extent and distribution in advanced cases (see Vol. I, p. 381). Stereoscopic plates are essential to the best results.

Prognosis in Chronic Ulcerative Phthisis.—When proper treatment is instituted in the stage of incipiency and rigorously carried out, the prospect of a cure is good. The frequency with which limited obsolescent, even healed tuberculous lesions are found post mortem in the lungs of individuals dead of other diseases or by accident, bears ample testimony to the intrinsic tendency to recovery. It has been said that more people recover from pulmonary tuberculosis without being aware that they have had it than die of it. *Treatment instituted while the diagnosis is still provisional justifies a hopeful prognosis—80 per cent. of recoveries; when the diagnosis has become certain the prognosis is grave—20 per cent. of recoveries.*

The prognosis in the advanced cases is unfavorable and in the far advanced cases hopeless. In individual cases, the early recognition of the disease, a good family history, limited local lesions, slight constitutional reaction, and the disposition and means to make a business of getting well are favorable conditions in the prognosis. The pleurogenous cases often run a slow and relatively favorable course. The cases characterized by recurrent blood spitting are unfavorable. Only in the worst cases is the course of the disease relentlessly progressive. As a rule there are periods varying from weeks to months in which the lesions remain quiescent, the symptoms subside, the general health improves, and there is a gain in weight. The duration varies from some months to several years, the average being about two and a half years. A remarkable decrease in the death-rate from consumption has taken place in recent years. This is to be ascribed to the discovery of the tubercle bacillus, the recognition of the fact that tuberculosis is an acquired rather than an inherited disease, and the dissemination among the people of the knowledge by which its spread can be restricted. Tuberculosis, the great scourge of mankind, has become since the work of Robert Koch (1882) the object of preventive medicine in its most advanced development—international and national associations and congresses, state and local anti-tuberculosis societies and conferences, sanitoria and dispensaries, popular courses of lectures and traveling exhibitions and museums.

(c) FIBROID PHTHISIS.

This term is used to designate a very chronic tuberculous process in the lungs, with relatively slight ulceration and much development of fibrous tissue. It may begin as a tuberculous bronchopneumonia or fol-

low an ordinary ulcerative phthisis. In a large proportion of the cases it begins as a chronic tuberculous pleurisy. The anatomical and clinical condition is practically that of pulmonary cirrhosis. One or both lungs may be affected; if both, one to a much greater extent than the other. There are vomicæ at the apex, surrounded by dense fibroid tissue, and bronchiectatic cavities elsewhere. The pleura is greatly thickened, and encapsulated cheesy masses, with patches of recent tubercle, and enlarged bronchial glands are present. Amyloid disease of the liver, spleen, and intestines develops in the advanced cases.

Symptoms.—Cough, often paroxysmal and more common in the morning, purulent expectoration, sometimes fetid, and dyspnœa upon exertion constitute the symptom-complex. Blood spitting occurs. The patients are thin, but frequently have fair health. Fever is not a common symptom.

Physical Signs.—The chest is flat, the shoulders lower, and the clavicles prominent. The vocal fremitus is diminished. Resonance is greatly impaired and has the tympanitic quality. At the apex cavernous, at the base bronchial, respirations are heard. Râles are not common, but coarse moist and gurgling râles may be present when fluid collects in the cavities or dilated bronchi. The superficial area of cardiac dulness is increased, the impulse may be seen and felt in two or more interspaces, and the heart is displaced toward the affected side.

Diagnosis.—DIRECT.—This rests upon the symptoms and physical signs as detailed above.

DIFFERENTIAL.—The distinction between tuberculous and non-tuberculous pulmonary cirrhosis cannot in all cases be made *intra vitam*. The presence of tubercle bacilli in the sputum is positive. When not found during life they are often present in the lesions after death. Atrophic emphysema presents some points of resemblance to fibroid phthisis, but differs from it in being a symmetrical affection and occurring only in aged persons.

Prognosis.—The outlook is favorable as to life, unfavorable as to recovery. The disease is chronic, lasting from ten to twenty or twenty-five years. The patient is usually able to attend to his affairs. There are cases characterized by recurrent hemorrhages, and death sometimes occurs from hæmoptysis.

Turban has suggested the following scheme for uniform records for comparative statistics in tuberculosis of the lungs:

1. Stadium = extent and severity of disease in the lung
   - I. Disease of slight severity, affecting at most one lobe or two half lobes.
   - II. Disease of slight severity, more extensive than I, but affecting at most two lobes; or severe, and affecting at most one lobe.
   - III. All cases of greater extent and severity than II.
2. Disease quiescent or progressing
3. Length of time since onset
   - To date from first occurrence of symptoms, such as persistent cough, hæmoptysis, or pleurisy.
4. General condition
   - Satisfactory.
   - Unsatisfactory.
5. Digestion
   - Normal.
   - Abnormal.
6. Pulse-frequency
   - To be taken in morning during repose.
7. Temperature
   - Daily maxima over 101.3°.
   - Daily maxima between 99.7° and 101.3°.
   - Temperature normal with two-hourly rectal temperature (mouth temperature 0.4°–0.5° lower).
8. Tubercle bacilli and mixed infection
   - Tubercle bacilli present.
   - Tubercle bacilli absent.
   - Mixed infection.
9. Tubercular complications
   - Name of affected organ.
10. Other complications
    - Name of disease: serious complications such as heart disease, nephritis, or diabetes are to be noted:
11. Capacity for work
    - Full, undiminished.
    - Slightly reduced.
    - Much reduced or lost.
12. Result of treatment
    - Improved.
    - Not improved.
    - Died.

Nos. 1–10 are filled up on admission or commencement of treatment. Nos. 11 and 12 on discharge.

XXXII. SYPHILIS.

Lues.

Definition.—A chronic specific infectious disease, caused by the Treponema pallidum. It is propagated by inoculation and characterized by, (a) a peculiar initial lesion—the chancre; (b) constitutional symptoms with mucous and cutaneous lesions and enlargement of the superficial lymph-nodes; (c) the development of granulomatous lesions in the various tissues of the body and in some cases after a long interval; (d) the evidences of parasyphilis—dementia paralytica or tabes dorsalis. These effects of the infection are consecutive, and constitute (a) the primary stage, (b) the secondary stage, (c) the tertiary stage, (d) the parasyphilitic or paraluetic stage. Syphilis is frequently transmitted from the parent to the child—hereditary syphilis.

Etiology.—Predisposing Influences.—Syphilis is a venereal disease and is usually acquired by illicit sexual intercourse. When acquired by an innocent person in the marital relation or by accidental means, it is described as *syphilis insontium*. Individual susceptibility is universal and affects all periods of life. Accidental infection is common among medical men. The fingers are usually the site of the primary lesion. Chancres upon the lip or tongue may result from the conveyance of the virus by kissing, the use of drinking utensils, the pipe, and other indirect methods. The infection is active in the oral and pharyngeal lesions—mucous patches. Unnatural vices are responsible for a certain proportion of the primary sores about the mouth. A nurse suckling a syphilitic infant may be inoculated upon the nipple and is also liable to accidental inoculation upon the lip, finger, or elsewhere. Local epidemics of syphilis among infants from arm-to-arm vaccination have been reported. Such accidents are no longer possible. Syphilis has been transmitted in tattooing. Hereditary transmission may take place from the father, the mother presenting no evidence of infection—*sperm inheritance, paternal heredity.* In rare instances a child begotten by a father in the active—secondary—stage has shown no evidence of syphilis. In equally rare cases the child of a father who, after thorough treatment, has shown no signs of the disease has developed congenital syphilis. There are usually unknown factors in problems of this nature. After vigorous systematic treatment, and the lapse of three years after the entire disappearance of symptoms, a man may be allowed to marry and is not likely either to infect his wife or to beget infected children.

Transmission from the mother is called germ inheritance—*maternal heredity.* A woman suffering from syphilis in the active stage is liable, when conception occurs, to bear a syphilitic child. As a rule both parents are syphilitic, the one having infected the other. A very remarkable fact is set forth in Colles's law, which, briefly stated, is this: A child that is affected with hereditary syphilis, its mother showing no signs of the disease, will not infect the mother. Such a child will infect its nurse or others, but the mother appears to have acquired an immunity without manifesting any of the usual phenomena of the disease. In the case of the mother

becoming infected after conception, the child may show the signs of congenital syphilis or, less frequently, it may escape. A parent or parents in the stage of tertiary syphilis may have non-syphilitic children.

Exciting Cause.—The spirochæta described by Schaudinn in 1905, and named by him *Spirochæta pallida*, is the cause of the disease. This organism is very delicate, closely coiled, having pointed ends, and motile. A larger spiral organism found in association with it upon the surface of syphilitic sores, and also upon the ulcerated surfaces of non-syphilitic lesions, and in smegma from healthy men and women, he named Spirochæta refringens. S. pallida—*Treponema pallidum*—is present in syphilitic lesions at various stages of the disease, as well as in the organs of congenital syphilis and the placenta, and in greater numbers in the active lesions. It has been found in the syphilitic lesions of inoculated monkeys and rabbits. It has been grown in culture by Noguchi.

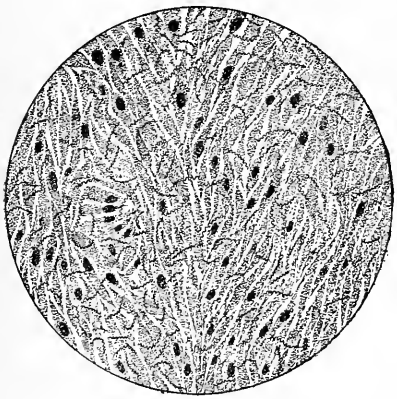


Fig. 281.—Treponema pallidum in a chancre.

(a) ACQUIRED SYPHILIS.

The primary and secondary and the secondary and tertiary stages often overlap or merge into each other.

The Primary Stage.—A period of incubation varying from two to four weeks, exceptionally longer, elapses between the inoculation and the appearance of the initial sore. This lesion consists of a small red papule, which gradually enlarges and breaks down in the centre, forming a circumscribed, superficial ulcer, with a peculiar hard, gristly, or cartilage-like movable base, which still further increases in size and is known as the indurated or hard chancre. This initial lesion may remain small and readily elude observation when just within the urethra or in the female genitalia. In other localities it usually appears as a conspicuous and characteristic sore. It is usually single; exceptionally multiple. In the course of a week or two the associated lymph-nodes undergo a painless and indolent enlargement. A little later the epitrochlear and retrocervical lymph-nodes become enlarged. This lymphadenoid hyperplasia is always bilateral and it attains its maximum in the course of eight or nine weeks.

The Secondary Stage.—The earliest indications of constitutional infection are usually manifest within a period varying from six to twelve weeks. They consist of the following phenomena: *Fever,* usually so mild as to attract little attention, 101° F. (38.5° C.), sometimes marked, less frequently severe. In type it may be subcontinuous, remittent, or intermittent; in duration indefinite, sometimes only subsiding upon the vigorous use of antisyphilitic treatment. It may not appear until late in the

course of the disease. The recognition of syphilitic fever is of great diagnostic importance. It may simulate malaria or the symptomatic fever of advanced pulmonary tuberculosis, or hectic fever due to other causes.

Anæmia.—The erythrocytes often fall rapidly to 3,000,000 and occasionally lower. Leucocytosis is infrequent. There is pallor with a sallow or muddy tinge of the skin. Slight hæmatogenous jaundice sometimes occurs in consequence of the rapid destruction of the erythrocytes. The superficial lymph-nodes, especially the suboccipital and epitrochlear glands, remain enlarged and tender. Lassitude, headache, rhachialgia, and the vague pains of a general infection are common. The designation syphilitic cachexia has been applied to cases in which this symptom-group is pronounced.

Cutaneous Lesions—Syphilodermata.—The earliest eruption is usually macular or roseolar. The individual spots are irregularly oval, of large size and often run together. They are symmetrically distributed upon the trunk and anterior surfaces of the arms and thighs. In color they are reddish-brown, often so faint as to be scarcely observed, sometimes vivid or coppery. This exanthem usually fades in the course of some weeks, but sometimes recurs at subsequent periods in the course of the disease. Later a papular eruption may appear upon the face and trunk, not unlike acne. This syphilide occasionally appears upon the forehead just below the edge of the hair—*corona veneris.* Frequently associated with it is a pustular eruption, suggestive of the variolous rashes. This combination of papules and pustules appearing upon the head and trunk, especially when syphilitic fever is present, may give rise to an erroneous diagnosis of smallpox. Still later squamous rashes appear, much like psoriasis but less scaly, coppery in color, and often confined to the palms and soles. Papulosquamous lesions are by no means rare. These eruptions frequently appear in the above order, but sometimes in a different succession, and two or more are often present at the same time. Symmetry and polymorphism are characteristic of the syphilodermata in the secondary stage. *Flat Condylomata.*—About the vulva and anus, upon the perineum, at the corners of the mouth, occasionally at the umbilicus, and in the folds of the armpits and groins, and elsewhere where the skin is constantly moist or there are opposing cutaneous folds, there sometimes appear, but not in all cases, flat warty growths, slightly elevated, with distinct borders and a moist, grayish surface. The secretion causing these lesions is inoculable and they are in the highest degree characteristic of syphilis. *Alopecia Syphilitica.*—Not rarely the hair, and often the eyebrows and lashes, fall out during the secondary stage. The loss of hair may be in patches, like those of alopecia areata, or there may be a general thinning. *Onychia Syphilitica.*—The nails are, in some cases, affected by a syphilitic inflammation involving the matrix, and are lost or become deformed.

Lesions of the Mucous Membranes.—The oral mucosa is chiefly affected. About the time of the appearance of the rash the throat and mouth become sore. There is a general erythematous angina, more intense than elsewhere, upon the tonsils and pharynx, where are frequently visible small, superficial ulcers with well-defined, scalloped borders and grayish-white

surfaces—*mucous patches*. These patches are also common on the tongue, the lips, and the buccal mucosa. They are characteristic of syphilis and the secretion from their surface is highly inoculable. Whitish patches upon the tongue—*leucomata*—sometimes occur, especially in smokers. Papillary hypertrophy of the mucosa about the vulva or at the verge of the anus may give rise to warty excrescences of considerable size—*condylomata*.

Other lesions of diagnostic importance are iritis, which is often encountered early in the secondary stage and tends to recur; much less frequently choroiditis and retinitis, and deafness from otitis media or labyrinthine disease. Abortion and miscarriage are common and repeated interrupted pregnancies are very suggestive. Periostitis is a border-line lesion marking the late secondary or early tertiary stage. It especially involves the tibiæ, clavicles, cranial bones, and less frequently the sternum. It is usually circumscribed and often associated with nodes. Upon palpation the surface of the long bones is rough, and nodular bosses may be felt upon the bones of the skull. There is tenderness upon pressure, and pain, which is usually worse at night.

The Tertiary Stage.—There is no distinct time between the secondary and tertiary stages. Tertiary lesions are sometimes present shortly after infection; sometimes they make their appearance along with the phenomena which are characteristic of the secondary stage; more commonly they do not appear until a longer or shorter, often a remote, period after the lesions of that stage have subsided. The third stage of syphilis is characterized by certain lesions of the skin, the development of gummata, disease of the bones, and amyloid degenerations. *Cutaneous Lesions.*—Circumscribed nodular lesions are common. They appear in groups, which are irregular, asymmetrical, and characterized by the formation of deep, rounded ulcers which involve the deeper layers of the skin and tend to coalesce, healing at one point and spreading at another, and leaving deep scars as they heal. Rupia, a deep ulcerating tertiary lesion covered by stratified, oyster-shell-like crusts, is much less common than formerly.

Gummata.—These lesions are circumscribed and vary in size from minute bodies to tumors sometimes reaching five centimeters in diameter. They develop in the skin, subcutaneous tissue, mucous membranes, internal organs, muscles, and bones. In the bones they form dense, hard, hemispherical, subperiosteal masses called nodes. Gummata of the skin and subcutaneous tissue tend to break down and form deep ulcers, which heal slowly and leave deep, disfiguring scars. Under treatment they are frequently absorbed. Gummata of the mucous membranes are especially common in the mouth, nose, and pharynx. They involve underlying structures and often give rise to extensive and deep ulceration and necrosis of cartilage and bone. Perforation of the nasal septum, destruction of the nasal bones, perforation and more or less extensive destruction of the hard and soft palates, and adhesions of the uvula or soft palate to the pharyngeal wall are common effects. Ulceration and necrosis of the cartilages of the larynx also occur. Stricture of the rectum is one of the results of gummatous infiltration and ulceration. Syphilomata are common in the internal organs. They sometimes form agglomerations of large size. Their usual

FIG. 281.—Tertiary syphilis (gummata).—Atlas of Clinical Medicine (Dr. Byrom Bramwell). By courtesy of the author.

course is to undergo fibroid metamorphosis with puckering and deformity. Syphilitic aortitis is the most common cause of isolated aortic insufficiency and aortic aneurism. Syphilitic nodes and periostitis have already been described. Further lesions are extensive and deep necrosis, which may become perforating, as in the bones of the cranium, the formation of exostoses which may cause serious and obscure pressure symptoms, as in the brain or spinal cord or the articulations. Syphilitic dactylitis, often followed by permanent deformity, is the manifestation of gummatous infiltration and periostitis of the bones of the fingers and toes. Much less common are gummata of the muscles and myositis syphilitica. Amyloid degeneration is common in syphilis even in the absence of chronic suppuration. It occurs especially in neglected cases of the acquired disease and is rare in the congenital form.

The Parasyphilitic Stage.

After an interval of years and in many instances after a long absence of luetic symptoms, there develop insidious, usually progressive and always grave evidences of disease of the cerebral cortex—*paresis* or *dementia paralytica* (q. v.) or of the spinal meninges and roots of the spinal nerves—*Tabes dorsalis* or *locomotar ataxia.* (q. v.)

(b) HEREDITARY SYPHILIS.

The infant may show the characteristic symptoms at birth or may present the appearance of health. In the latter case the evidences of infection appear in the course of one or two months.

1. **Symptoms at Birth.**—The child is undersized and usually wasted and wrinkled. Bullæ may be present on the wrists and ankles and scaly patches upon the palmar and plantar surfaces. Mucous patches upon the nasal and oral mucous membrane, rhagades at the angles of the mouth and at the anus are characteristic. There is enlargement of the liver and spleen. Nodular thickening of the bones and, in some cases, separation of the epiphyses occur. Such children snuffle, are extremely feeble, are difficult to feed, and usually perish within a short time. Hemorrhage is occasionally encountered. It is more common at the navel but may be subcutaneous, or there may be bleeding from the mucous surfaces.

2. **Early Symptoms.**—When born without symptoms, syphilitic children are often plump and well nourished and remain so until some time between the third and eighth weeks. The earliest symptoms are usually those of a syphilitic endorhinitis, namely, impeded nasal respiration, difficulty in nursing, snuffling, and a mucopurulent, sometimes bloody discharge. In severe cases necrosis of the nasal bones may occur, followed by characteristic deformity of the face. Involvement of the Eustachian tubes and middle ear results in deafness. Such cases constitute one of the groups of deaf-mutes. Cutaneous lesions appear about the same time. They consist of a certain general muddy sallowness, in sharp contrast to the fresh rosy skin of a healthy infant, patchy erythema or eczema, or large, irregular coppery patches with well-defined borders. These erup-

tions are frequently first seen upon the buttocks, but may invade other regions. A papular syphilide is common. Mucous patches develop and are highly contagious. The secretion from these lesions usually constitutes the means of infection in wet-nurses and others. Alopecia, onychia, dactylitis also frequently occur as the case goes on. A general adenopathy is not common, but the lymph-nodes in relation to local lesions of the skin often undergo an indolent enlargement. The spleen is enlarged; the liver less constantly and to a less extent. The large relative size of the liver in the new-born is to be kept in mind.

3. **Later Symptoms.**— Children suffering from congenital syphilis may regain the appearance of health under judicious management. Very frequently they remain undersized and badly nourished and look prematurely old. The facies and cranial development are very often characteristic. The skull is frequently asymmetrical, the forehead prominent, the bridge of the nose in some cases depressed, the lips pouting, with radiating linear scars, especially at the corners of the mouth. At the second dentition and at puberty the symptoms of hereditary syphilis frequently reappear.

Hutchinson Teeth—Notched Teeth.—The upper central incisors are peg-shaped, shorter, and narrower than normal, and especially narrower at the cutting edge than at the neck. The enamel is usually well formed, not pitted and thinned as after prolonged non-specific sickness in infancy, and there is at the cutting edge a single notch of varying depth in which the enamel is deficient and the dentin exposed.

Other symptoms are interstitial keratitis, iritis, deafness of labyrinthine origin, bone lesions, and, in particular, a gummatous periostitis which gradually causes marked thickening and deformity and which shows an especial tendency to affect the tibiæ. The nodes of late hereditary syphilis are usually symmetrical and are sometimes mistaken for rickets. They may first appear in adolescence. There may be enlargement of the spleen and visceral gummata.

The question of the transmission of syphilis to the third generation remains undecided.

(c) VISCERAL SYPHILIS.

1. **Syphilis of the Central Nervous System** (see Diseases of the Nervous System).

2. **Syphilis of the Lung.**— Pulmonary syphilis is a very rare condition. The following forms are described: (a) *White Pneumonia of the Fœtus.*— The process may involve extensive portions of a lobe or an entire lung. The affected tissue is heavy, airless, and of a grayish-white color. The alveoli are filled with desquamated epithelium and their walls are thickened and infiltrated. (b) *Gummata* irregularly scattered throughout the lung, especially in connection with the bronchi and more abundantly about the root than elsewhere. There is an associated bronchopneumonia. (c) *Fibrous interstitial pneumonia* beginning at the root of the lung and extending along the bronchi and vessels. This sclerotic process may begin in the pleura and involve the connective-tissue framework, especially in the

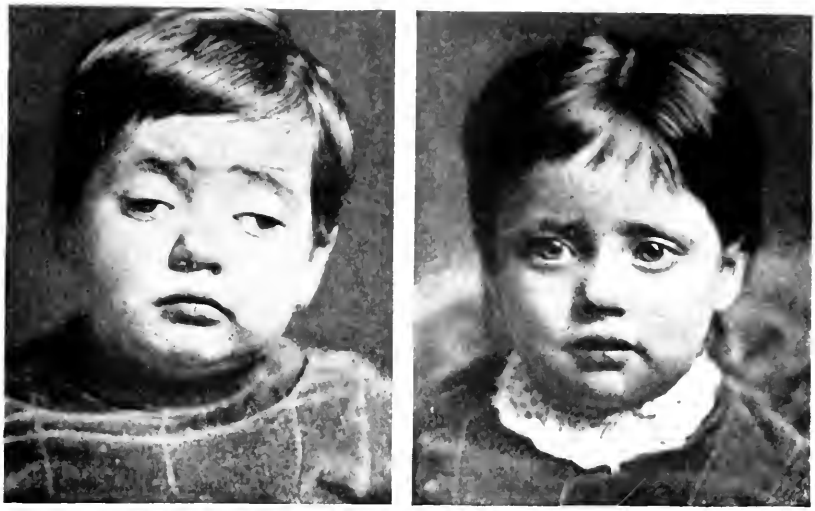


Fig. 281*b*.—Ophthalmoplegia externa before and after treatment

Fig. 281*c*.—Hand from the case of ophthalmoplegia externa represented in Fig. 281*b* before and after treatment.—Atlas of Clinical Medicine (Dr. Byrom Bramwell). By courtesy of the author.

interlobar tissue. It principally affects the portions of the lung adjacent to the root. It is encountered in individuals with a syphilitic history or in whom there are other forms of visceral syphilis, and is sometimes associated with gummata. As in other forms of pulmonary sclerosis, bronchiectasis is often present.

Symptoms.—The clinical manifestations are those of pulmonary tuberculosis or pulmonary sclerosis. In the former case the absence of tubercle bacilli upon repeated examination, and the absence of signs of destructive lesions, as elastic tissue, are suggestive; in the latter the signs of chronic interstitial pneumonia and of bronchiectasis are present. The acute syphilitic pneumonia and chronic syphilitic phthisis of French authors are not generally recognized as clinical entities. Other signs of syphilis are present.

3. **Syphilis of the Liver.**— The following forms are described: (a) *Diffuse Syphilitic Hepatitis.*—This is common in congenital syphilis. The organ is large and firm and shows the presence of minute and larger gummata and extensive connective-tissue hyperplasia. (b) *Gummata.*—In congenital syphilis gummata of various sizes may occur at any period. In the acquired disease they are usually among the later manifestations of the acute process. They are commonly multiple and may attain the size of an orange. They undergo fibroid changes with contraction and cause remarkable deformities of the organ; in rare cases softening takes place with the formation of one or more fluctuating tumors. (c) *Syphilitic Perihepatitis.*—Glisson's capsule and the connective tissue along the portal canals are thickened. Great vein obstruction may occur when the connective-tissue proliferation extends along the large venous trunks. (d) *Amyloid Liver.*—This change is very common in syphilis. Gummata may be present or a consecutive diffuse hepatitis may occur.

Symptoms.— The clinical phenomena are by no means constant. Congenital syphilitic hepatitis can scarcely be diagnosticated with precision even when suspected. The organ is enlarged and firm. There may be jaundice. In the adult the symptom-complex of atrophic cirrhosis is frequently present. The symptoms are sallowness or slight jaundice, digestive disturbances, loss of weight, and ascites.

Irregularity in the outline of the liver dulness occurs in many of the cases. The evolution and involution of gummata cause progressive and retrogressive deformities of the liver which are of great importance in diagnosis. These syphilitic tumors are less dense in consistence than the surrounding tissue in hepatic or amyloid disease, and can in some cases be differentiated from it upon palpation. In syphilitic perihepatitis an audible and palpable friction rub may sometimes be recognized; jaundice is present in one-third of the cases and may be intense. Pains in the hepatic region occur and the signs of ascites and of splenic enlargement are by no means rare. In amyloid liver the symptoms of amyloid disease in other organs are usually present. The liver is enlarged, smooth, and firm. Its outline may be irregular. There is commonly also enlargement of the spleen. Anæmia, polyuria with albumin and casts, and a tendency to dropsy are present.

4. **Syphilis of the Digestive Tract.**—The œsophagus and stomach are very rarely involved. Ulceration of the small intestine is likewise uncommon. The rectum is far more often affected. Rectal syphilis is more common in women. The lesions are due to gummata in the submucous tissue above the internal sphincter, which undergo ulcerative changes which become chronic and on healing cause stenosis. There may be tenesmus, discharge of bloody pus with the stools, and pain on defecation. Later the symptoms are those of stenosis.

5. **Syphilis of the Circulatory System.**—The Heart.—Except in the aortic valve-system valvular lesions are infrequent. Both vegetations and gummata have, however, been observed. Mural lesions are common. They comprise gummata, fibroid induration, amyloid degeneration, and endarteritis obliterans. Changes in the blood-vessels of the heart occur both in the congenital and the acquired disease. Valvular lesions give rise to definite murmurs. Syphilis of the myocardium may be present without symptoms; those characteristic of myocarditis are usual. Sudden death may occur. The Stokes-Adams phenomenon may be symptomatic of syphilitic disease of the interventricular septum. Gummata have been found post mortem.

The Arteries.—There are two forms of syphilitic arteritis, an obliterating endarteritis which is not distinctive, and a gummatous periarteritis which involves especially the smaller arteries of the brain and the branches of the coronary arteries, and is specific. Syphilitic changes in the arteries are etiologically related to arteriosclerosis and aneurism. Syphilitic aortitis is most common in the later stages of the disease. Its usual seat is the root of the aorta. The clinical manifestations relate to insufficiency of the aortic valves; aneurism, and implication of the coronary valves. The probability of its occurrence should be considered early in the course of every case of syphilis.

Syphilis of the cerebral arteries at the base of the brain may lead to partial obliteration with softening or atrophy or to the development of aneurismal dilatation, with characteristic phenomena—tumor symptoms.

6. **Syphilis of the Kidneys.**—Gummata and amyloid degeneration constitute the common changes. The former cannot be recognized *intra vitam;* the latter presents the usual symptoms. An acute syphilitic nephritis, without specific characters, has been described.

7. **Syphilis of the Testicles.**—Gummata in the substance of the testis is not uncommon. It may be mistaken for tuberculosis. It is usually painless and does not tend to invade the skin or to undergo softening or suppuration. An interstitial orchitis may develop as a slowly progressive affection unattended with pain and resulting in induration and atrophy. One testis is usually affected to a greater extent than its fellow.

Diagnosis.—1. General Diagnosis of Syphilis.—(a) The Primary Lesion.—The surgeon is more frequently consulted than the medical man. In a suspicious sore the following points are of importance: a history of exposure within a month or six weeks; induration; movability; sluggish ulceration; scanty secretion; slight painless enlargement of the inguinal glands. These traits belong equally to the chancre upon the genitalia and

elsewhere. The history of exposure may, however, be in default. The patient may prefer to conceal the actual fact at the risk of his future health as well as his character for veracity; or the inoculation may have occurred in marital intercourse or otherwise by non-genital infection. The initial lesion may not have attracted the patient's attention. This is especially liable to occur in women. In man the lesion is sometimes inconspicuous and may be mistaken for preputial herpes or an abrasion; or it may be masked by coincident chancroids, or, when at the meatus or in the urethra, by a gonorrhœa, or finally an extragenital chancre, even when well characterized, may fail to arouse the suspicion of the practitioner as to its true nature. There is only one diagnostic rule, namely, to preserve a guarded and discreet openness of mind in all doubtful cases and carefully watch for subsequent developments. A negative anamnesis is without value.

(b) The Secondary Stage.—Consecutive events are important. The history of exposure, especially when doubtful, and the history of a subsequent sore, however doubtful, are of great diagnostic value. Sore throat and roseola are usually the first symptoms which attract the patient's attention. A painful erythematous angina, with tonsillar ulceration and mucous patches, with a symmetrical, faint, brownish-red macular rash upon the trunk, and painless enlargement of the inguinal, suboccipital, and epitrochleal lymph-nodes, especially when associated with fever, constitutes a symptom-complex upon which a direct diagnosis may be made. Later polymorphous rashes, corona veneris, alopecia, irregular fever, and anæmia are confirmatory.

The Wassermann Test.—This serum test requires a very careful technic and is exposed to many sources of error. It therefore should only be undertaken by trained laboratory workers. The reliability of this diagnostic method has been fully established. Wassermann has given the results of 3000 tests, of which 1010 were upon cases surely non-syphilitic and used as controls. In 1982 syphilitic cases about 90 per cent. gave positive results. In cases without manifest symptoms at the time, "latent syphilitis," about 50 per cent. gave positive reactions. It is, however, not only a means of diagnosis but it is also a therapeutic guide. In the course of treatment a positive reaction may be absent with entire lateney as to symptoms, only to reappear again, either with or without symptoms. The intensity corresponds to some extent to the activity of the process. A positive result is common in cases of tabes and paresis. Cholesterinized antigen appears to be preferable to the alcoholic extract of syphilitic liver.

Provocative Test.—When the Wassermann reaction is negative in a suspected case a small dose of salvarsan is given and in a short time the Wassermann is repeated. The theory is that the salvarsan acting upon the lesions causes the spirochætes to enter the circulation.

Luetin Test (Noguchi).—This procedure consists in the superficial injection into the skin of one arm of a suspension of *Spirochæta pallida* in pure culture and devitalized by heat, the other arm being similarly treated by an uninoculated control medium. Separate syringes are used for the luetin and the control. The amount of luetin employed for a single test is 0.07 c.c. A positive reaction consists in the appearance, some hours later

or upon the following day, of an inflammatory nodule surrounded by a bright red areola which is fully developed in 48 to 72 hours. Mild constitutional symptoms may occur. Pustulation sometimes appears. This test is of use in tertiary cases, but rarely yields a positive reaction in primary or untreated secondary syphilis.

(c) The Tertiary Stage.—The anamnesis is here also of great importance. It is often defective. Sometimes discretion suggests a very guarded investigation of the past history of the patient. Inquiry should be made concerning persistent rashes and falling of the hair. Careful inspection of the throat and skin should be made for the signs of past lesions. Scars in the groins are insufficient evidence. Suppurating buboes are usually due to chancroids, not syphilis. Slowly progressive ulcerating lesions of the skin, advancing in one direction and healing in another—serpiginous—gumma and gummatous ulceration, perforation of the nasal septum, of the hard or soft palate, necrosis of the nasal or cranial bones, the signs of iritis, the presence of nodes, irregular periosteal thickening or exostosis, especially upon the clavicles, tibiæ, or bones of the skull, particularly when two or more of them are associated, constitute diagnostic data of final importance in the direct diagnosis. But these lesions are often wholly absent and the sufferer from tertiary syphilis, and particularly the sufferer from nervous syphilis, may be entirely free from the gross or visible external manifestations of the disease. In many cases of nervous syphilis it is impossible to elicit a history of marked secondary signs.

In syphilis of the cerebrospinal system the cell count of the cerebrospinal fluid, an increase in the globulin content and a positive Wassermann reaction in this fluid are of diagnostic value (see Serology).

(d) The Fourth or Paraluetic Stage.—Neurological considerations, lumbar puncture with positive findings in the cerebrospinal fluid and a positive Wassermann reaction of this fluid dissipate all doubts as to the diagnosis.

The Wassermann reaction is of the highest diagnostic importance. It is positive in every stage of syphilis except the period of development of the chancre, and during and for a period after treatment by salvarsan or other efficient measures. There are few exceptions to this general rule and no cases can be regarded as cured until one or more negative reactions have occurred at a considerable interval after treatment.

2. **Diagnosis of Hereditary Syphilis.**—Repeated miscarriages are, in connection with any of these phenomena, of diagnostic value. The efflorescence of the characteristic rash associated with snuffles, mucous patches, and rhagades within the first three months justifies the direct diagnosis. At subsequent periods of life the characteristic facies, infantile development, symmetrical nodes, notched teeth, and interstitial keratitis tell their own story and may solve the problem of diagnosis in obscure nervous or visceral disease.

3. **Diagnosis of Visceral Syphilis.**—The anamnesis and the presence of the signs of former lesions are of primary importance. In the male careful search should always be made for vestiges of the primary sore. It is to be remembered that the manifestations of visceral syphilis are

usually not in themselves different from those of lesions due to other pathological processes, and that their true nature can be recognized only by the history of infection, the presence and association of characteristic external phenomena, the therapeutic test, and the Wassermann test.

1. *Syphilis of the Brain and Cord* (see Diseases of the Nervous System).

2. *Syphilis of the Lung.*—The clinical diagnosis of this rare condition in any of its forms can seldom be made with precision. Chronic interstitial pneumonia with the signs of bronchiectasis, or chronic bronchopneumonia in a person with a history of syphilis or presenting well-characterized lesions in other parts of his body, may be of syphilitic origin. Tuberculosis of the lungs and gummata may coexist.

3. *Syphilis of the Liver.*—The diagnosis is most important, as it is essential to the choice of treatment. The irregularly enlarged liver, with soft circumscribed gummata, may suggest cyst, abscess, or malignant tumor. Under such circumstances an unnecessary surgical operation might be performed. A history of infection, collateral lesions, and fair general health suggests syphilis. The diagnosis in gummata forming large conglomerate tumor masses in the right or left lobe in absence of collateral evidence must remain obscure. In cirrhosis and perihepatitis recovery under specific treatment is often the only sign. Irregularity of outline, which, when there is ascites, can only be determined after paracentesis, is very suggestive. Amyloid disease of the liver is commonly associated with similar visceral changes elsewhere.

4. *Syphilis of the Digestive Organs.*—The history affords presumptive evidence in disease of the œsophagus. Syphilis of the stomach cannot be positively diagnosticated. The chronic course of syphilis of the rectum, the symptoms of gradual stenosis of the gut, and the results of digital examination by which a firm fibrous annular contraction is usually felt, quite unlike the irregular, ragged surface of ulcerating cancer, are essential diagnostic criteria.

5. The clinical diagnosis of *syphilitic disease of the heart and arteries* must be a provisional one. Cardiovascular changes, in no respect differing in symptomatology from those occurring in syphilitic subjects, are often due to other causes.

6. The diagnosis of *renal syphilis* cannot be made during life. Recent syphilitic infection, rapid development of renal symptoms, a high grade of albuminuria and marked œdema are suggestive. The urinary sediment should be examined for spirochætes.

7. *Syphilis of the Testes.*—The recognition of syphilis in these organs may be of great importance in obscure visceral disease. Syphilis, tuberculosis, and cancer are to be differentiated. Gummata involve the body of the testicle and give rise to irregular conglomerated masses, unattended with pain and showing no tendency to invade the skin or undergo softening. Tubercle more commonly affects the epididymis and is often associated with the signs of tuberculous disease elsewhere. Malignant disease runs a more rapid course, is attended with pain, and tends to involve the skin and undergo ulceration.

Therapeutic Diagnosis.—Treatment by salvarsan reinforced by mercury and the iodides under suitable conditions will frequently cause the disappearance of symptoms. Intermittent courses of treatment, repeated during long periods of time, usually prevent the recurrence of symptoms, arrest the tendency to abortion, and may be followed by the birth of healthy children in whom neither the early nor the late manifestations of syphilis occur. Obscure skin eruptions fade if of syphilitic origin.

With reference to visceral syphilis the following facts are important: The symptoms of nervous syphilis may, in early cases, disappear after the use of antisyphilitic remedies; in pulmonary syphilis the results are inconclusive; syphilis of the liver in certain of its forms is amenable to treatment and in some case the improvement is rapid and permanent; syphilis of the heart and arteries shows retardation rather than cure and here the therapeutic test is useless. The symptoms of the secondary stage yield promptly, while in the tertiary stage the gummatous lesions yield more or less gradually and the sclerotic lesions are but slightly if at all influenced. Parasyphilitic diseases are sometimes benefited by antisyphilitic treatment. Finally there are rare cases of acute malignant syphilis which run a rapidly fatal course wholly uninfluenced by treatment.

Prognosis.—Under early, systematic treatment, repeated from time to time in courses of proper duration for a period of three or four years, an apparent cure is, in the majority of cases, established and maintained.

XXXIII. GONORRHŒA.

Definition.—A contagious catarrhal inflammation of the genital mucous membrane, chiefly propagated by impure sexual intercourse, and due to the gonococcus of Neisser.

Etiology.— This organism is a coffee bean or biscuit shaped coccus occurring free and in pairs as diplococci in the protoplasm of the leucocytes in gonorrhœal inflammation. It is present in the pus of gonorrhœal[1] urethritis and conjunctivitis and the exudate of gonorrhœal endocarditis and arthritis. In smears stained with alkaline methylene blue the organisms are usually intracellular. It is Gram negative.

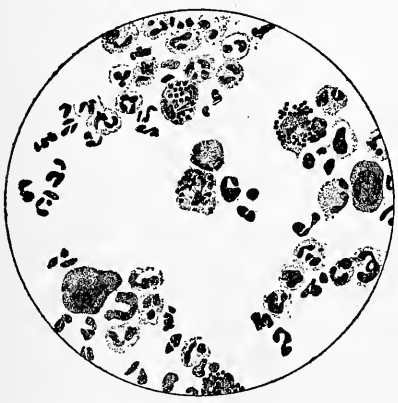


Fig. 282.—Spread of pus containing gonococci.

This wide-spread venereal infection is scarcely inferior in importance to syphilis. In truth, when we take into consideration the facts that syphilis is much less virulent than formerly in its early and late constitutional effects, and that it gives rise to symptoms which compel the most ignorant and inexperienced to seek professional advice at a period when it is still amenable to treatment, and that gonorrhœa, while retaining all its capacity for immediate and late harmfulness, is too often regarded as a trifling local

disorder, the very existence of which may be unsuspected by the female patient, we may even question whether gonorrhœa is not the more serious disease of the two.

The gonorrhœal infection may limit itself to the mucous membrane of the genitalia—(1) the primary local infection; it may invade the genito-urinary organs by direct continuity of structure—(2) secondary local infection; or finally, it may be swept into the blood stream and give rise to (3) constitutional infection.

With reference to the spread of the infection in the genito-urinary tract, it is a question of extent. Every case in the male is at first an infection of the anterior urethra. With reference to systemic effects, it is a question of degree; any case is liable to systemic disturbance, and malaise, feverishness, head and back pains, and other symptoms of constitutional infection are often present at the onset or later in the course of the attack. The fever associated with the initial symptoms is due to the absorption of toxins.

The Primary Local Infection.—The gonococcus may persist in a slight urethral discharge capable of giving rise to the disease for long periods after the patient has thought himself cured; thus innocent women frequently become infected upon marriage. It is also to be remembered that the existence of a urethral stricture bears a causal relation to cystitis, pyelitis, persistent rhachialgia, lassitude, general disability, and neurasthenia, and in acute disease it may be the cause of urinary retention. The ocular infections—*ophthalmia neonatorum* and *gonorrhœal conjunctivitis*—are extremely dangerous and if neglected frequently lead to loss of sight.

Symptoms of Gonorrhœa in the Male.—The period of incubation is from one to three days; in mild cases it may be longer. The attack begins with a burning sensation at the meatus with redness and swelling, soon followed by pain, micturition and a mucous discharge, which soon becomes purulent and abundant and contains gonococci. Flocculi and shreds are seen in the urine. In many cases painful nocturnal erections occur—*chordee*. The duration of untreated cases varies from three or four to several weeks. In chronic cases the discharge gradually becomes thin and scanty—*gleet, goutte militaire*. When the inflammatory process invades the posterior urethra, pain and tenderness in the perineum, tenesmus, frequent micturition, and often blood at the close of the act as determined by the three glass test (*q. v.*) are present. Marriage should be forbidden until urethral shreds composed of pus cells, and gonococci in smears of the urethral secretion have failed to be present for a period of several months.

Gonorrhœa in the Female.—The disease may be acute or chronic. The acute stage may pass unnoticed or be wholly absent. Men apparently free from gonorrhœa frequently transmit it to their wives. Prostitutes are as a rule infected. The sites of primary infection are in order of frequency the urethra, cervix uteri, vulva and vagina, the vulvovaginal glands with retention cysts or abscess-formation. In ascending gonorrhœa the endometrium, the tubes and ovaries, and in rare cases the peritoneum are involved. The symptoms vary greatly in intensity. They may be trifling or consist of intense burning sensations in the external genitalia and vagina, painful micturition, tenesmus and an abundant, yellowish

discharge. The vulva in severe cases is swollen, red, tender and from the orifice of the urethra there may be expressed a drop or two of pus containing gonococci. Infection of the adnexa is usually attended with pelvic pain, local thickenings, abscess formation and sterility and often renders surgical operation imperative.

Ophthalmia Neonatorum.—Infection of the child's eyes may occur during labor. The free use of a one per cent. solution of silver nitrate or a twenty per cent. solution of argyrol may prevent intense and destructive gonorrhœal conjunctivitis.

Secondary Local Infection.—The gradual extension of the specific inflammation from the mucous membrane primarily involved gives rise, in the male, to posterior urethritis, epididymitis, prostatitis, periurethral abscess; in the female to abscess of the vulvovaginal glands, metritis, salpingitis, inflammation of the ovary, and, in rare cases, to acute peritonitis; in both sexes to cystitis and pyelitis, usually mixed infections.

Constitutional Gonorrhœal Infection.—Gonorrhœal Sepsis—Septicopyæmia.—The presence of the gonococcus in the blood has been demonstrated. There are cases of rapidly fatal general infection, usually associated with suppurative lesions in the urinary tract. The symptoms are chill, high temperature of irregular range, profuse sweats, muttering delirium, and stupor deepening to coma.

Cardiac and Articular Localizations.—Gonorrhœal Endocarditis.—This localization is of frequent occurrence in gonococcus septicæmia. Gonococci have been isolated from the blood during life, and the vegetations upon the valves after death. The endocarditis is often of the malignant type. In a majority of the cases the endocarditis has followed an arthritis, but it may occur in the absence of the joint affection or the latter may follow it. Pericarditis may occur, and an acute gonorrhœal myocarditis has been observed.

Gonorrhœal Arthritis.—The designation gonorrhœal rheumatism is erroneous and misleading and should be abandoned. This localization has been observed in infants in connection with ophthalmia neonatorum and in young children in whom gonorrhœa is common as the result of accidental infection by towels or clothing, or of vicious practices. It is most common between twenty and thirty. Males suffer from the joint affection more frequently than females. It may occur at any time during the course of the urethral discharge. Most commonly the arthritis begins during the acute stage and is followed by a subsidence of the discharge, which is, however, usually only temporary. It may occur after discharge has greatly diminished or not for several weeks after the beginning of the attack. A single joint may be affected; more commonly two or even three are involved. Polyarthritis is rare and the migratory form characteristic of acute rheumatism does not occur. The joints remain inflamed and only slowly get well. The ankles, knees, and wrists are especially liable to gonorrhœal inflammation and the temporomaxillary, sternoclavicular, vertebral, and sacro-iliac articulations are frequently attacked. The inflammation is endo- and periarticular. In the latter case the exudate sometimes extends along the sheaths of the tendons. The effusion into the joints is that of a serous synovitis. It may be seropurulent. The gonococcus may be iso-

lated from the endo- and the periarticular exudates. In some cases the results are negative. Mixed infections—staphylococci, streptococci—occur.

Gonorrhœal arthritis tends to become chronic. Relapses are frequent; disorganization or disabling ankylosis may result. Among the complications are iritis, endocarditis, pericarditis, and pleurisy.

Diagnosis.—Direct.—The anamnesis is most important. The existence of a mucopurulent urethral discharge is not positively conclusive. A young person suffering from gonorrhœa may also contract rheumatic fever. In doubtful or obscure cases the complement-fixation test must be employed. In the female a vaginal discharge should be examined for gonococci. Clinically, a limited number of joints involved, the persistence of the inflammation without migration, the association of endocarditis of the malignant form, and implication of the temporomaxillary, sternoclavicular, or vertebral articulations are of diagnostic value.

Differential.—*Rheumatic Fever.*—The evanescent—*migratory*—character of the arthritis, the number of joints involved,—*polyarthritis*,—the frequent rapid and complete resolution of the inflammation, a history of exposure to cold or wet, of previous attacks, of heredity, and the commonly lower intensity of an endocarditis, when present, are in favor of rheumatic fever. *Pyæmic or Septic Arthritis.*—The presence of a suppurative focus, caries, osteomyelitis with toxæmic phenomena, as irregular chills, high fever of irregular remittent or intermittent type, profuse sweating, rapidly developing anæmia, the implication of a single joint or, at most, two or three, and the evidences of large intra-articular effusion, are in favor of septic arthritis. *Gout.*—If the patient be a middle-aged male, energetic and self-indulgent, the attack be sudden, the great toe, ankle, knee, or wrist involved, the swelling tense, the surface livid and glossy, and tophi be present around the small joints or in the helix of the ear, a diagnosis of gout may safely be made. I have seen a calculus impacted in the urethra cause a free purulent discharge, followed by an attack of gout. *Arthritis Deformans.*—There are cases in which, for a time, the joint affection, whether arthralgic, polyarthritic, or monarthritic, cannot be distinguished at the time of acute exacerbations from gonorrhœal arthritis. No question arises in regard to the differential diagnosis except in cases of recent or chronic urethritis in the male or leucorrhœa in the female.

Prognosis.—Gonorrhœal infection in its systemic form is always a serious matter. The virulent septic cases prove fatal in a few days or a week or two; the endocarditis frequently assumes the malignant type; the arthritis is commonly rebellious to treatment and not rarely is followed by lasting, even permanent disability. Salicin and the salicylates are useless.

XXXIV. EPHEMERAL FEVER—FEBRICULA—SIMPLE CONTINUED FEVER.

Definition.—Fever of short duration, occurring in the absence of definite lesions or known specific cause, and characterized by elevation of temperature and the derangements of function which commonly attend it. Fever lasting twenty-four or forty-eight hours and ceasing completely is designated *ephemeral fever;* an attack of three or four days' duration, *febricula;* and one lasting a week or more, *simple continued fever.*

Etiology.—Children and neurotic individuals are more liable to transient febrile attacks than others—a fact due to the instability of the heat-regulating mechanism. Several groups of cases are described: (a) symptomatic, (b) toxic, and (c) infectious.

(a) In many cases the fever is doubtless due to unrecognized local lesions, as in angina tonsillaris or catarrhal bronchitis of the larger tubes, with little or no cough and no râles; or to slight injury such as results from a fall upon the head.

(b) Among the toxic cases are those which arise from indigestion or gastro-intestinal catarrh with the absorption of fever-producing substances. The fever sometimes follows prolonged mental or physical effort, exposure to damp and cold without definite lesions, exposure to the sun but not to a degree sufficient to cause thermic fever, and the inhalation of the concentrated emanations from decomposing organic matter. There are instances in which a number of persons have been at the same time taken ill, with nausea, vomiting, fever, and, in some instances, collapse symptoms after being present at a very offensive post-mortem examination or the opening of an obstructed sewer or of a mortuary vault.

(c) Mild or abortive cases of the infectious diseases have been regarded as febricula. Stillé called attention to the occurrence of cases of cerebro-spinal fever so mild that they can "only be recognized by the lurid light of the epidemic." The true nature of the mildest form of enteric fever—typhus levissimus—may be readily overlooked, and there are cases of scarlet fever, measles, and rheumatic fever without distinctive symptoms beyond a transient fever for two or three days—larval cases of the infectious diseases. In view of these facts, it is evident that the more closely the cases of transient fever without obvious symptoms are studied, the fewer will be encountered that are really neither symptomatic nor specific, and there is a tendency to do away with this group of fevers altogether as a nosological entity. On the other hand, every practitioner occasionally encounters cases for which no other place in the classification of diseases can be found.

Symptoms.—The onset is usually abrupt; exceptionally gradual with lassitude and languor. The usual symptoms of febrile infection are present. In rare instances, especially in children, there may be chilliness or a convulsion. Defervescence takes place by crisis between the second and the fourth days; if later, usually by rapid lysis.

A direct **diagnosis** may be made from the abrupt onset of the fever, its short course and the critical termination in the absence of local lesions and cutaneous rashes. The **prognosis** is invariably and essentially favorable.

XXXV. ROCKY MOUNTAIN SPOTTED FEVER.

Tick Fever.

Definition.—An acute infectious endemic disease, prevalent in the northwestern mountainous regions of the United States, and due to the bite of a tick. It is characterized by the gradual onset of symptoms common in the acute infections, a papular or petechial eruption, and an irregular fever of variable duration terminating by lysis.

This disease, long known under designations such as "mountain fever," "black fever," and "spotted fever," has recently been made the subject of scientific investigation under the auspices of the Montana State Board of Health (1902), the Public Health and Marine Hospital Service (1903), and the American Medical Association (1906–1907), and is now recognized as a nosological entity.

Etiology.—Predisposing Influences.—Certain districts in the Rocky Mountains are the exclusive regions in which the disease occurs. The great majority of the reported cases have been observed in April, May, and June A few have occurred in March and July. No period of life affords exemption. The disease has been noted as early as the second and as late as the seventy-fourth year. Ranchmen, lumbermen, engineers, and prospectors have supplied the greater number of cases.

Exciting Cause.—Despite various hypotheses, the infecting organism has not yet been demonstrated. It is inoculated by the bite of a tick—*Dermacentor andersoni*. Tick fever may be caused in monkeys and guinea-pigs by the inoculation of the defibrinated blood of human beings suffering from the disease. Some observers have attributed the disease to the inoculation of the infecting principle by mosquitoes.

Symptoms.—The average duration of the period of incubation appears to be about seven days. The onset is attended by nausea and vomiting, muscular pains, malaise, headache, and epistaxis, chilliness or a distinct chill. The temperature rises abruptly to 103°–104° F. (39.5°–40° C.) and its range may be subcontinuous or intermittent. At the expiration of from sixteen to twenty days defervescence takes place, followed by subnormal temperatures for a few days. In fatal cases the temperature usually remains high, but a preagonistic fall sometimes occurs. The eruption shows itself about the third or fifth day, first appearing upon the ankles, wrists, and forehead, and gradually spreading over the whole body. It remains, however, more abundant upon the extremities than upon the trunk. It is at first maculopapular and disappears upon pressure. In the course of a few days it becomes more distinctly papular and does not disappear upon pressure or when the skin is made tense. Many of the spots become petechial and in the severer cases more or less extensive ecchymosis occurs. The intervening skin is congested, slightly cyanotic, and jaundiced. In favorable cases the eruption fades with the defervescence and its disappearance is followed by desquamation. Gangrene of the penis or scrotum or of the toes has been reported. In mild cases the rash may not appear. Gastro-intestinal symptoms are prominent. The liver and spleen are enlarged. The urine is diminished in volume, of high color and specific gravity, and contains albumin and casts of all kinds. The blood shows a moderate secondary anæmia and in some cases a moderate leucocytosis—13,000.

Diagnosis.—The direct diagnosis rests upon the endemic occurrence of a disease having the above symptom-complex in particular localities and at certain seasons of the year, a history of tick or mosquito bites, sudden onset, chilliness or high continued or intermittent fever, the appearance of the maculopapular eruption upon the third, fourth, or fifth day, petechiæ, ecchymosis, and defervescence followed by desquamation.

Prognosis.—Rocky Mountain spotted fever is a very fatal disease. According to several observers the mortality varies from 70 to 90 per cent. In some seasons the cases are mild and most of them have terminated in recovery. Among the phenomena which in individual cases render the outlook especially unfavorable are deep jaundice, extensive hemorrhage into the skin, delirium, and exhaustion.

XXXVI. ICTERUS INFECTIOSUS.

Weil's Disease; Acute Febrile Icterus.

Definition.—An acute infectious disease characterized by sudden onset with chill, followed by high fever and jaundice.

Etiology.—Predisposing Influences.—The clinical picture suggests the severer cases of catarrhal jaundice and various febrile forms of gastro-intestinal disease that may run their course with or without jaundice. It corresponds very closely to the disease observed by Griesinger in Cairo, and Kartulis in Alexandria, and described under the designation *bilious typhoid* or *typhus biliosus*. Many cases reported as Weil's disease clearly do not belong to that category, such as santonin poisoning, septicæmia, abortive enteric fever, and the so-called hepatic form of enteric fever. The view that Weil's disease is a form of rheumatic fever complicated by a resorption icterus is not generally accepted. Some etiological considerations support the assumption that the disease is the manifestation of a specific infectious process. Among these are the following: The cases which correspond to Weil's description usually occur sporadically, but not rarely they appear in groups in circumscribed localities, and during the hot season. Males are more affected than females—90 per cent. Certain occupations exert a predisposing influence, butchers, tanners, and laborers in sewers being especially liable to the disease. It has been attributed to the drinking of contaminated water, and epidemics, especially among soldiers, have been ascribed to the swallowing of such water during bathing. The disease is most frequent between the twenty-fifth and the fortieth years of life. It is uncommon in childhood, and rare after fifty.

Exciting Cause.—The recent work of Japanese investigators has demonstrated the fact that Weil's disease is a *Spirochætosis*. The infecting organism is *S. icterohemorrhagica* and the carrier the rat.

Symptoms.—The attack begins abruptly, usually without prodromes and often with a chill. Headache, vertigo, pains in the back and limbs occur. There is great lassitude. The temperature rises rapidly to 104° F. (40° C.) or higher, and is remittent in type. It lasts from eight to fourteen days. There are recurrences of the fever, and in a considerable proportion of the cases relapses occur. Stupor and delirium occur, and the resemblance of some of the cases to enteric fever may be striking—a resemblance increased by the early development of splenic enlargement. Jaundice makes its appearance between the third and fifth days and is of variable intensity, being in a considerable proportion of the cases deep and attended by clay-colored stools. The liver is increased in size and tender upon pressure. The urine is commonly albuminous, with hyaline and epithelial casts, and

sometimes contains red blood-corpuscles. Hæmaturia is not very uncommon. In the fatal cases deep stupor, delirium, and coma precede death. There is rapid wasting during the attack. Muscular pains persist after the defervescence and are among the last symptoms to disappear. Angina tonsillaris is occasionally an early complication. Herpes facialis and other cutaneous lesions, as erythema and hemorrhage into the skin, have been observed. A group of the graver cases are hemorrhagic. Parotid bubo is a rare complication. The duration of the attack varies from two to four weeks, and the convalescence is slow.

Diagnosis.—The direct diagnosis rests upon the occurrence of jaundice with the symptoms of an acute severe infection, quite unlike ordinary catarrhal icterus on the one hand, and without the phenomena of the specific infections on the other. The age, sex, and occupation of the patient are to be considered. It is probable that some of the local epidemics reported as catarrhal jaundice of severe type have been outbreaks of Weil's disease.

Prognosis.—The death-rate is low, most of the cases terminating in recovery.

XXXVII. GLANDULAR FEVER.

Definition.—An infectious disease of children, characterized by sudden onset, erythematous angina, enlargement of the tonsils, high fever of short duration, and swelling and tenderness of the lymph-nodes of the neck, particularly those along the posterior border of the sternocleidomastoid muscles.

Etiology.—Predisposing Influences.—Certain individuals, particularly in childhood, exhibit a peculiar irritability of the superficial lymphatic glands, some of which become enlarged and painful in almost any disease, however trifling. Enlargement of the lymph-nodes is characteristic of measles, rötheln, tuberculosis, syphilis, and many other diseases, and, when acute, is commonly attended with some degree of fever. Glandular fever—*Drusenfieber of Pfeiffer*—is a definite nosological entity, the predisposing influences to which, except that it is almost exclusively a disease of childhood, are wholly unknown. It occurs sporadically and in epidemics.

The exciting cause has not yet been determined.

Symptoms.—The onset is sudden, with a rise of temperature to 101°–103° F. (38.5°–39.5° C.). There is pain on moving the head and neck, together with nausea, vomiting, and abdominal pain. The angina is not as a rule intense nor of long duration. The adenopathy shows itself upon the second or third day, the glands becoming enlarged in varying degree to the size of a walnut, painful and tender to the touch. There is slight periglandular œdema, but no general swelling or redness, and but little difficulty in swallowing. In a limited proportion of the cases substernal uneasiness and an unproductive, paroxysmal cough are the signs of implication of the tracheal and peribronchial glands. The axillary, inguinal, and mesenteric glands are sometimes involved. The fever is of short duration but the enlarged glands only slowly undergo involution. Suppuration has been noted.

Diagnosis.—The direct diagnosis rests upon the presence of the foregoing symptom-complex. The differential diagnosis must be made by exclusion.

Prognosis.—Recovery is the rule. The occurrence of suppuration in some of the affected glands, otitis media, retropharyngeal abscess, and severe, even hemorrhagic nephritis may protract the illness or cause it to terminate in death; but these accidents are infrequent.

XXXVIII. MILIARY FEVER.

Sweating Sickness.

Definition.—An acute infectious disease characterized by fever, profuse sweating, and an eruption of miliary vesicles.

Miliary fever, or the sweating sickness, prevailed extensively in England in the fifteenth and sixteenth centuries. Outbreaks have occurred within recent years in France, Italy, and Austria. They have sometimes extended over wide areas; more frequently they have been limited to districts or villages. These epidemics have lasted in some instances three or four weeks; in others they have run their course in a week or ten days. When the disease shows itself it attacks, like influenza and dengue, a large number of persons in a very short time.

Etiology.—Nothing is known of the pathogenic principle which gives rise to miliary fever.

Symptoms.—After prodromal symptoms of short duration, the attack sets in suddenly with moderate fever, profuse sweating, and epigastric distress. These symptoms are followed by an erythematous rash and the copious eruption of miliary vesicles, most abundant upon the neck and trunk. In the more severe cases the symptoms of an intense infection—high fever, profound depression, hemorrhage, and delirium—are present. Desiccation and desquamation occur. The duration of the disease varies from one to four weeks.

Diagnosis.—Direct.—This rests upon the development of an acute illness, characterized by the above symptoms, in a large proportion of the inhabitants of a locality in rapid succession, and the absence of the symptoms of influenza, dengue, or other infectious maladies.

Differential.—The sweating might suggest malaria, but the orderly succession of chill, fever, and sweat which characterize the ague fit in the regularly recurring forms, the well-defined periodicity, the effects of quinine, and the presence of Laveran's parasite in the blood would settle any doubt. Influenza bears a strong resemblance to miliary fever. Its pandemic prevalence, the prominence of catarrhal symptoms, the frequency of high fever, and the absence of the vesicular rash determine the question of diagnosis. Dengue differs from miliary fever in its geographical distribution, being a disease of tropical and subtropical climates; in its mode of onset and course; in its early arthropathy; in its eruptions; and in the fact that profuse sweating and a copious vesicular eruption are uncommon. Rheumatic fever might suggest miliary fever by the abundant perspirations which are common; but the prominence and migratory character of the arthritis, the higher fever, and the sporadic or endemic occurrence would at once dispel any uncertainty in regard to the diagnosis.

Prognosis.—In the more malignant forms death occurs in the course of several hours. The mortality in the early course of epidemics is high. In some of the recent outbreaks the death-rate has been low.

XXXIX. FOOT-AND-MOUTH DISEASE.

Aphthous Fever.

Definition.—An acute infectious disease of cattle, sheep, and pigs, but also met with less frequently in other domestic animals, characterized by fever, salivation, and a vesicular eruption upon the mucous membrane of the mouth, nose, and conjunctiva, less frequently also upon that of the vulva and upon the udder and teats. In the sheep, goat, and pig the affection manifests itself particularly about the hoof, while in the horse only the oral mucous membrane is involved. This disease, which occurs in widespread epizoötics, is readily transmissible to man, and numerous epidemics have been described. It is more common as a sporadic affection.

Etiology.—Predisposing Influences.—All those occupations which involve contact with animals suffering from the disease, their fodder or manure, or the stables in which they are housed, predispose to the disease.

Exciting Cause.—The disease is caused by the filterable virus of Loeffler-Frosch. Its presence in the contents of the vesicles and in the saliva, milk, and urine, has been demonstrated by intravenous and intraperitoneal injection. It retains its virulence in stalls, fodder, and dung heaps for a period of several months, and is capable of transportation by fomites. The most common source of infection in man is by the raw milk of infected animals. Herbwig's experiments upon himself and his assistants demonstrated this mode of transmission beyond doubt. The cream also contains the virus, as well as butter, curds, and cheese made from the infected milk. The possibility of acquiring the disease by handling the meat of infected animals is to be considered, though prolonged cooking may render it innoxious as an article of food.

Symptoms.—In man, after an incubation period of from three to five or eight days, the disease begins with lassitude and pains in the head, back, and limbs. These symptoms are associated with dryness of the mouth, difficulty in swallowing, and nervous symptoms, such as vertigo and insomnia. Complete loss of appetite, vomiting, and fever ensue. The last is, however, by no means a constant symptom. In some cases there is nose-bleeding. If the infection has occurred by way of the oral mucosa there are seen, in the course of a day or more, vesicles which come out in successive crops and are preceded by a more or less diffuse and very painful inflammation. In the course of a short time the vesicles increase in size and then rupture, leaving superficial ulcers which gradually heal with scars. There is salivation, great pain in eating, and a fetid breath. Urticaria and roseolous and scarlatiniform rashes appear, and in some cases crops of vesicles resembling the vesicles upon the mucous membrane of the mouth. The last gradually undergo desiccation and healing without ulceration. When infection takes place by way of lesions upon the skin, constitutional symptoms develop first, vesicles appear near the seat of infection, the above-described rashes follow, and sometimes, but by no means invariably, the vesicles subsequently develop in the mouth.

Diagnosis.—Direct.—This rests upon the presence of the foregoing symptoms in an individual who has been exposed to the danger of

contact with infected animals or their surroundings, or of eating infected food products. In a doubtful case, experimental inoculation should be practised and for this purpose the calf, on account of its greater susceptibility, should be selected.

Differential.—The ordinary form of aphthous stomatitis is not attended by the constitutional symptoms of a severe infection, nor accompanied by cutaneous rashes. The vesicles run their course so rapidly that they are seldom seen prior to the formation of the circumscribed painful ulcers, with sharp edges and yellow bases, to which they give rise. The acute exanthemata may be suggested by the constitutional symptoms and the buccal and cutaneous eruptions. Varicella, however, usually lacks the fever and lassitude of foot-and-mouth disease; measles is characterized by catarrhal symptoms not present in the affection, and scarlet fever by a diffuse non-vesicular angina and generalized erythema developing within forty-eight hours after an abrupt onset. Diphtheria, which may be suggested by the appearance of the ulceration in the mouth in certain cases, reveals its pathological identity by the Klebs-Löffler bacillus. In any suspicious case the etiological factor in diagnosis is important.

Prognosis.—In a majority of the cases in adults recovery takes place in two or three weeks. Owing to the difficulty in taking food, and the serious nature of the gastro-intestinal derangements in children, the outlook is far more serious, and progressive emaciation and debility are frequently followed by death. Septic infection by way of the mucous and cutaneous lesions may prolong the illness.

XL. ERYSIPELOID OF ROSENBACH.

Erythema Migrans; Erythema Serpens.

Definition.—An erythematous inflammation of the fingers, due to the inoculation of an, as yet, undetermined pathogenic principle, associated with putrescent animal matter, and characterized by swelling, tension, dark red or purplish discoloration, well-defined edges, and a tendency to advance from the point of origin, with moderate pain, some itching and burning, and the absence of constitutional symptoms.

Etiology.—Predisposing Influences.—Erysipeloid is a comparatively rare disease. It has been especially studied by Rosenbach in Germany (1887), and Gilchrist (1904) and Jopson (1908) in this country. Occupation constitutes the most important predisposing influence. The affection occurs almost without exception among those who are engaged in handling dead animal matter under conditions which render it liable to putrefaction, as dealers in fish (especially shell-fish), game, and poultry, and butchers and cooks. Medical students, laboratory workers, and taxidermists are also liable to the infection. Those who handle crabs are especially exposed to the danger of contracting erysipeloid, which is sometimes spoken of as "crab cellulitis."

Exciting Cause.—Rosenbach and others have described a cladothrix, but subsequent investigators have failed to obtain such an organism. The manner of inoculation, the nature of the lesions, the constant clinical

course of the disease, and the pathological findings, which are those of an inflammation of the entire corium, and, to some extent also, of the subcutaneous tissue, are suggestive of a specific infection. No specific organism has, however, been demonstrated.

Symptoms.—The infection results from an injury to the skin. There may be a number of small wounds. The period of incubation varies from a few hours to several days. The inflamed skin is tense, livid, elevated above the level of the adjacent surface, and characterized by a sharply defined border which advances toward the hand and may invade the palm, the dorsum of the hand, and other fingers than the one first affected. The nose and cheeks are occasionally involved. As the border advances resolution takes place in the part first attacked. Suppuration does not occur nor do vesicles, as a rule, develop. Complete recovery takes place in a period varying from two or three to ten or fifteen days. Fever and other constitutional symptoms do not occur.

Diagnosis.—The direct diagnosis rests upon the foregoing characters and may usually be made without difficulty. The differential diagnosis from the cellulitis caused by pyogenic organisms rests upon the mode of infection in the latter, the occurrence of suppuration, and the presence of pus-producing bacteria in the exudate; and from erysipelas by the absence of constitutional symptoms and the trifling nature of the erysipeloid affection. The prognosis is uniformly favorable.

XLI. ERYTHEMA INFECTIOSUM.

In 1896 Escherich described a feebly contagious disease of childhood characterized by a rose-colored maculopapular rash with trifling subjective symptoms, among which a mild erythematous sore throat is mentioned. The incubation period varies from six to fourteen days and the rash appears first upon the face and consists of maculopapules, in some instances sharply marginate, in others gradually fading into the healthy skin. It disappears momentarily upon pressure. In the course of twenty-four hours it invades other parts of the body, sometimes presenting the appearance of urticaria. It is especially marked upon the extensor and outer surfaces of the extremities. It lasts from six to ten days, fading first upon the face, where it first appeared, and is not followed by desquamation. The superficial lymph-nodes are not enlarged. The attack does not confer immunity from scarlet fever, measles, or rubella. No outbreaks have been observed in America. The direct diagnosis rests upon the character of the rash and its distribution, the absence of constitutional symptoms, and mild transmissibility. The differential diagnosis from these diseases depends upon the absence respectively of their specific etiologic and clinical manifestations. Erythema infectiosum bears only the most remote resemblance to scarlet fever, measles, rubella, urticaria, and certain drug rashes.

II.

THE DIAGNOSIS OF DISEASES CAUSED BY ANIMAL PARASITES.

A. DISEASES DUE TO PROTOZOA.

i. Psorospermiasis.

This term is applied to the diseases produced by the sporozoa other than hæmospiridia—protozoa that only live parasitically in the cells, tumors, or organs of other animals. They are mostly cytozoa. This class includes Gregarinida and Coccidiidea. The latter is the cause of a disease common in the rabbit in which the liver is the seat of small whitish nodules, seen upon section to be dilatations of biliary ducts. Coccidia are found in the epithelial cells lining these cyst-like dilatations. Rainey's corpuscles or tubes are ovoid bodies containing sickle-shaped, unicellular organisms—Sarcocystis miescheriana—found within the sarcolemma of various animals, especially the hog.

Visceral Psorospermiasis; Coccidiosis.—Infection takes place by swallowing the spores or oöcysts containing spores. The gastric juice causes the spores to open and frees the sporocytes, which by way of the common duct reach the biliary ducts and penetrate the epithelial cells. A limited number of cases has been observed in man.

Symptoms.—Those of severe infection: fever (sometimes intermittent), dry tongue, nausea, diarrhœa, and tenderness over the liver and spleen, which are enlarged. The parasites have also been found in the ureters and kidneys. The diagnosis during life has not been made.

ii. Amœbic Dysentery.

Definition.—A colitis caused by Amœba dysenteriæ and characterized by pain, tenesmus, and frequent stools containing mucus and blood. Dysentery occurs as an acute and chronic disease. Liver abscess is common.

Etiology.—Predisposing Influences.—Dysentery is more prevalent in tropical countries. In Egypt and India it is endemic, frequently epidemic. It occurs sporadically in all temperate climates. It is a water-borne disease and infection may take place by drinking contaminated water, or eating raw vegetables washed with it.

Exciting Cause.—Amœba dysenteriæ, first described by Lambl in 1859, later by Lösch in 1875, is classed among the Rhizopoda. This organism frequently exhibits the differentiation between the hyaline ecto- and the granular endosarc very clearly, especially in the pseudopodia. It contains a vesicular nucleus and contracting vacuoles. Amœbæ are seen in great numbers in the stools, being found especially in the shreds of mucus or pus, in the pus of liver abscess and abscess in other positions which occasionally occur, and in the purulent expectoration in hepatic abscess discharging by way of a pulmonary fistula. An amœba frequently

contains red blood-corpuscles. They can be grown in cultures from the stools and intestinal ulcers but not readily alone, a symbiotic organism being required. Amœba coli has been found in the stools of healthy persons. There may be different varieties, of which some are non-pathogenic.

The lesions to which this organism gives rise are situated in the large bowel, sometimes reaching as high as the lower part of the ileum. They consist of an inflammatory infiltration of the submucosa, followed by necrosis and sloughing of the mucous membrane, which results in the formation of circumscribed round, oval, or irregular ulcers with overhanging borders. The base of the individual ulcer consists, according to stage of development, of the submucous, muscular, or serous coat of the gut. There is a remarkable tendency to extend by undermining the mucous membrane with the formation of deep, serpiginous ulcerating tracts or fistula. In severe cases the entire intestine is greatly thickened and extensively ulcerated. When the process is less extensive, the rectum, the hepatic and sigmoid flexures, and the cæcum are points of selection. Healing leads to extensive new formation of fibrous tissue and constriction of the bowel.

Fig. 283.—Amœba dysenteriæ.

The lesions of the liver consist of, (a) local necrosis of hepatic tissue in scattered patches, and (b) abscess formation. The abscess may be multiple and scattered throughout the organ, or single. The latter are usually situated in the right lobe and under the convex or diaphragmatic surface; less frequently near the concave intestinal surface. In the former case rupture frequently takes place into the lung or pleura. Less commonly, according to its location, the abscess may rupture into the inferior vena cava, pericardium, peritoneum, stomach, intestine, or the portal or hepatic vein. (See also Liver Abscess.)

Symptoms.—Acute Form.—This form is characterized by sudden onset, pain, tenesmus, frequent stools containing blood and mucus. Large sloughs may be passed. There is fever, not often intense. Rapid emaciation occurs and the patient may die in the course of a week or two. Hemorrhage of the bowels may take place or perforations with peritonitis. Recovery is, however, the rule. In many of the cases the disease becomes chronic. There is a moderate leucocytosis—9000 to 16,000. Eosinophilia is common.

Chronic Form.—The disease may be insidious in onset. There are subacute dysenteric attacks with pain, frequent stools, mucus and blood, and slight fever. These spells alternate with periods of constipation. The patients may have fairly good health, but are liable to indigestion, and the attacks are readily brought on by errors of diet, over-fatigue, sudden chilling and the like.

Diagnosis.—Direct.—The intestinal symptoms—pain, tenesmus, frequent stools with blood and mucus—justify a diagnosis of dysentery; the presence of amœbæ in the dicharges, a diagnosis of amœbic dysentery, alike in the acute and chronic cases. The diagnosis may be confirmed by the therapeutic tests—ipecac in salol coated pills or emetine by hypodermic injection.

Hepatic abscess may be entirely latent. More commonly there is enlargement of the liver at its upper or lower aspect with recurrent chills, fever, sweating, local pain, and œdema. The leucocytosis is high. With the establishment of a pulmonary fistula there is dark expectoration containing amœbæ.

Differential.—*Bacillary Dysentery.*—The diagnosis rests upon the absence of amœbæ and the agglutinating power of the blood-serum for the bacilli, and the more pronounced toxæmia. *Proctitis.*—Tenesmus and mucohemorrhagic stools may suggest dysentery, but the slight and transient nature of the attack and its manifest local character are of diagnostic importance.

Prognosis.—Many cases recover but recurrences occur. The mortality is about 25 per cent. Hepatic abscess adds to the gravity of the outlook.

iii. Trypanosomiasis.

Sleeping Sickness.

Definition.—A chronic disease caused in man by Trypanosoma gambiense and characterized by undulant fever, rapid pulse, weakness, loss of flesh, and frequently a protracted lethargy.

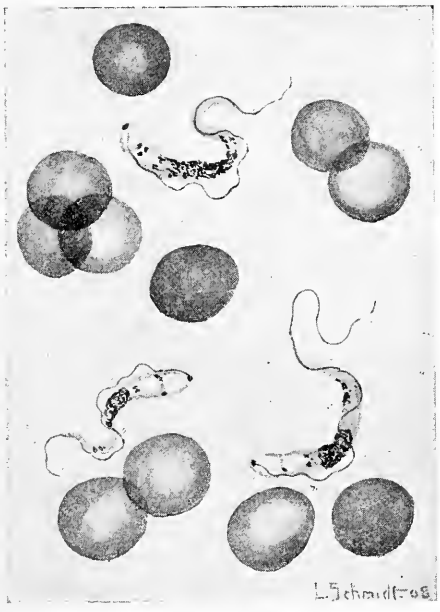


Fig. 284.—Trypanosoma gambiense.—After Bruce and Nabarro.

Etiology.—Predisposing Influences.—As a disease of horses and cattle trypanosomiasis is known as *surra* in India and the Philippines and is prevalent in South Africa. Africa has, in fact, no less than six trypanosomal diseases, all of them very important: nagana, dourine, galziekte, zouspana, Gambian horse disease, and human trypanosomiasis. Human trypanosomiasis is widely distributed in Uganda and the West Coast of Africa. It is conveyed by the tsetse fly—*Glossina palpalis.*

Exciting Cause.—This flagellate protozoön was first discovered by Gruby in the blood of the frog in 1843. The organism is a unicellular, elongated body having an undulating fold or membrane upon the dorsal edge which terminates in a flagellum of varying length. Stained specimens show a large nucleus and a small chromatin mass near one pole. It has recently been grown by Novy on artificial media in the labo-

ratory. It was later noted to be a common blood parasite in birds and fishes. It was found in 1878 by Lewis in the rat and subsequently in cattle and horses by Evans (1880) and Bruce (1895). Dutton first recognized trypanosomes in the blood of human beings in 1902. Trypanosomes have been observed with great frequency in the sleeping sickness or African lethargy and have a causal relation to that disease.

Symptoms.—As in the case of rats and other animals, trypanosome infection may be latent in human beings. Trypanosome Fever.—The symptoms consist of irregular fever, rapid pulse, weakness, swelling of the lymph-nodes and spleen, and œdema of the feet. This form of the disease has been produced in monkeys by inoculation. Sleeping Sickness.—The period of latency may be extremely prolonged, in some instances reaching five years. In a case of Manson's, symptoms developed in a fortnight. The early symptoms are dulness, apathy, headache, fever, difficulty in walking, tremor of the hands, and mumbling speech. The fever and drowsiness increase, the patient has to be aroused to take food and finally cannot be aroused at all. Death usually results from some complication, as septic meningitis.

Diagnosis.—Direct.—Exposure in an infected region, the history of an insect bite (especially by the African tsetse fly, Glossina palpalis), the varying but prolonged period of latency, the peculiar symptom-complex (especially the progressively deepening lethargy), and the presence of trypanosomes in the blood and cerebrospinal fluid are characteristic.

Prognosis.—Sleeping sickness is a very fatal disease. The duration after distinctive symptoms have occurred varies from three to twelve months.

iv. Dum-dum Fever.

Kala-azar.

Definition.—A chronic disease of the East due to a protozoön of the flagellate type and characterized by irregular fever, pulmonary congestion, anæmia, recurrent œdema of the feet, enlargement of the spleen and liver, and occasionally subcutaneous hemorrhages. This disease is identical with tropical cachexial fever and has been called Leishman-Donovan disease.

Etiology.—Predisposing Influences.—Dum-dum fever is prevalent in Oriental countries and occurs in Egypt. The European races rarely suffer.

Exciting Cause.—The bodies discovered by Leishman in 1900 and independently by Donovan in 1903, and regarded by Laveran and Mesnil as a new species of Piroplasma, have been found in the liver and spleen by puncture during life and also, in the majority of cases, in the circulating blood. They have been found in the mesenteric glands, intestinal ulcers, and bone-marrow. In stained smears of the fluid from the spleen and liver they appear as elongated oval or circular bodies with a spherical nucleus against the capsule and a rod-like body on the other side. They occur singly and in pairs and packed in phagocytic cells in the juice of the liver and spleen and in zoöglœa masses in the lung. Malarial parasites are not found.

Symptoms.—Constant enlargement of the spleen; usual but not invariable enlargement of the liver; moderate anemia; leucopenia; irregular fever prolonged for many months with occasional remissions; hemorrhages, subcutaneous and from mucous surfaces, especially the gingival and nasal mucosa; transitory œdemas, particularly of the legs and feet; and more or less marked pallor and pigmentation of the skin—these constitute the clinical picture. Dysentery and various secondary infections occur.

Prognosis.—Two forms of the disease are encountered, an intense form with recurrent attacks of high fever and rapidly developing cachexia, which terminates fatally in a few months, and a much milder form both as regards fever and cachexia, which runs a protracted course, death usually resulting from some intercurrent disease. The mortality, according to Leishman, varies from 70 to 96 per cent.

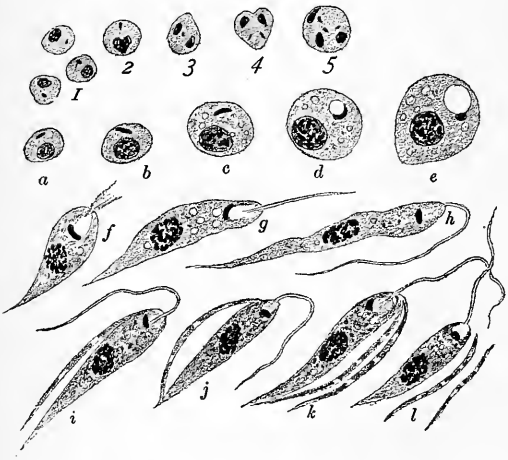


FIG. 285.—Leishman-Donovan bodies. 1, three parasites showing chromatin masses; 2, 3, 4, 5, parasites showing fission.—After Christophers. *a*, parasite from spleen; *b*, *c*, *d*, *e*, large parasites from cultures; *f*, *g*, *h*, bodies showing flagella; *i*, *j*, *k*, forms exhibiting unequal longitudinal fission; *l*, two spirillar forms separated from parasite.—After Leishman.

v. Malarial Fevers.

Definition.—A group of infectious diseases caused by the Hæmosporidia (Plasmodia) described by Laveran and transmitted to man by the bite of the similarly infected mosquito, comprising, (a) regularly intermitting periodical fever of tertian or quartan type, (b) irregular fever of remittent or continued type, and (c) a chronic cachexia and pernicious fever, often rapidly fatal, with anæmia and enlargement of the spleen.

Etiology.—PREDISPOSING INFLUENCES.—The *geographical distribution*, formerly wide, is becoming more restricted. The conditions necessary for the breeding of the mosquito, namely, heat and moisture, are everywhere predisposing factors to malaria. Regions of special prevalence are found in tropical and subtropical countries, as Panama, Central America, India, and Northern Africa, especially along the coasts and in the river basins. In Europe, Southern Russia and Lower Italy are still highly malarious. Germany and France are almost free, while the prevalence of the disease in Holland and England has practically ceased. In the United States the malarial fevers have steadily diminished since the colonial period. At the time of the first settlement of the country the prevalent fevers were malarial; as clearings were made and the soil tilled and drained, malarial fevers and enteric fever prevailed side by side until at length malarial fevers disappeared and enteric fever became predominant. Malarial fever has almost disappeared from New England and New York. It is now comparatively infrequent in Eastern Pennsylvania, New Jersey, and Maryland.

DESCRIPTION OF PLATE XIII.

THE PARASITE OF TERTIAN FEVER. (Drawn by Mr. Brödel for Thayer and Hewetson's paper, The Malarial Fevers of Baltimore, Johns Hopkins Hospital Reports, Volume V. We copy the original legend.)

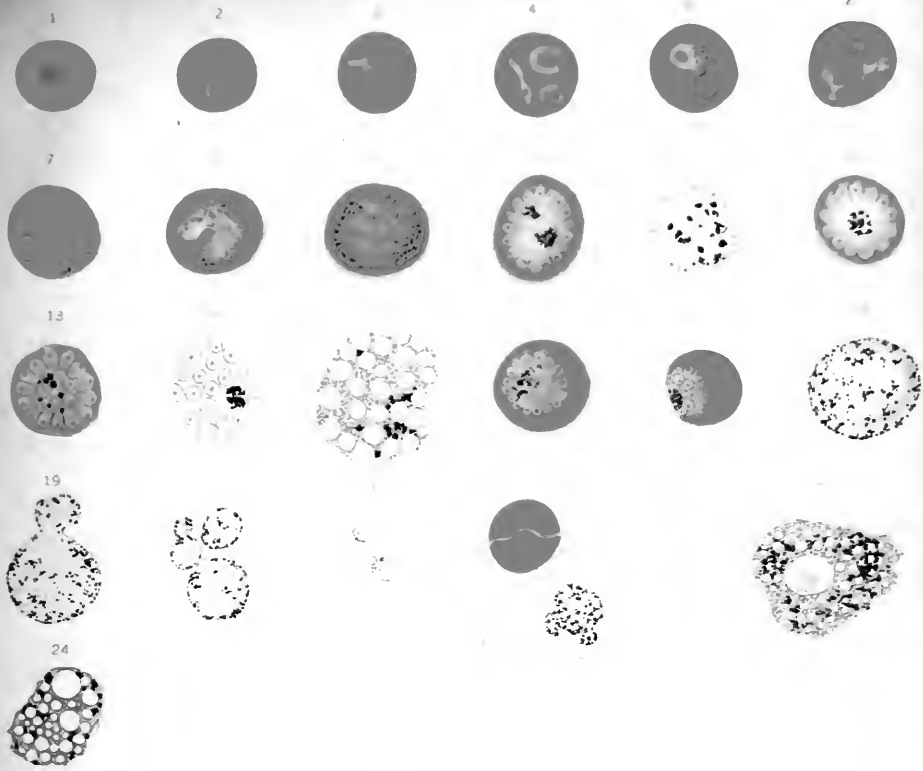
1. Normal red corpuscle.
2, 3, 4. Young hyaline forms. In 4, a corpuscle contains three distinct parasites.
5, 21. Beginning of a pigmentation. The parasite was observed to form a true ring by the confluence of two pseudopodia. During observation the body burst from the corpuscle, which became decolorized and disappeared from view. The parasite became, almost immediately, deformed and motionless, as shown in Fig. 21.
6, 7, 8. Partly developed pigmented forms.
9. Full grown body.
10–14. Segmenting bodies.
15. Form simulating a segmenting body. The significance of these forms, several of which have been observed, is not clear to the writers, who have never met with similar bodies in stained specimens so as to be able to study the structure of the individual segments.
16, 17. Precocious segmentation.
18, 19, 20. Large swollen and fragmenting extra-cellular bodies.
22. Flagellate body.
23, 24. Vacuolization.

THE PARASITE OF QUARTAN FEVER.

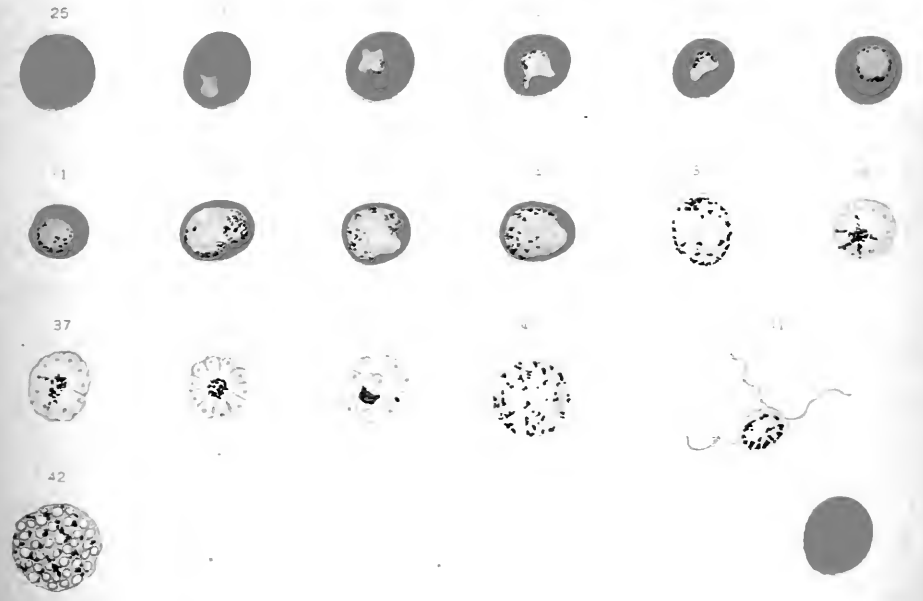
25. Normal red corpuscle.
26. Young hyaline form.
27–34. Gradual development of the intra-corpuscular bodies.
35. Full grown body. The substance of the red corpuscle is no more visible in the fresh specimen.
36–39. Segmenting bodies.
40. Large swollen extra-cellular forms.
41. Flagellate body.
42. Vacuolization.

The Parasite of Tertian Fever

PLATE XIII.



The Parasite of Quartan Fever



M Brockel fec

It still prevails in many regions of the South, especially on the Gulf Coast. The Northwestern States, the Pacific Coast, and the regions north of the St. Lawrence are practically free from it.

Season.—In the tropics the maximum prevalence corresponds to the rainy season, the minimum to the dry. In temperate climates there are a few cases in the spring, usually relapses—*vernal intermittent*. The greater number of cases occur in the early autumn—primary infection.

Locality.—Genera of the subfamily Anophelinæ, the only mosquitoes in which the malarial parasite of man develops, are distinctly rural insects, breeding in small, shallow pools and stagnant waters, in contradistinction to Culicinæ, which prefer human habitations and cities and deposit their ova in tanks and cisterns. Hence malarial diseases prevail more extensively outside of cities.

Exciting Cause.—To Laveran, a French Army surgeon, is due the credit of having discovered in 1880, in Algiers, in the blood of patients suffering from malarial fever, the hæmatozoön which he recognized as parasitic and regarded as the cause of the disease. A great number of observers have contributed to our knowledge of the subject. Among the most important steps in the development of that knowledge are the following: that the febrile paroxysm coincides with the sporulation or segmentation of a group of parasites; that the tertian, quartan, and pernicious fevers are due to different parasites; that infection takes place by the bite of the mosquito, species of the subfamily Anophelinæ; that the infecting mosquito must itself be previously infected by the blood of an individual suffering from malaria; and that the malarial parasites of man require two different hosts for their complete development—the asexual cycle taking place in the blood of man, the intermediate host, and the sexual cycle in Anophelinæ, the definitive host. So far as our present knowledge of the life history of the malarial parasites goes they exist only in the mosquito and man.

The parasite belonging to the class Sporozoa, order Hæmosporidia, has received many different names. It was designated *Plasmodium malariæ* by Marchiafava and Celli, and this term, although unsuitable according to the rules of zoölogical nomenclature, has remained in general use.

The Parasite in Man.—*Schizogonous Cycle.*—Three species are recognized, differing morphologically and in the form of fever which they cause. They are (a) the tertian (Plasmodium vivax), (b) the quartan (Plasmodium malariæ), and (c) the estivo-autumnal (Plasmodium immaculatum).

Tertian Parasite (*Plasmodium vivax*).—This species is the cause of tertian fever. Its cycle of development occupies forty-eight hours. It appears first in the red blood-corpuscles as a round or irregular unpigmented body which gradually increases in size. In the course of a few hours it has become ring-shaped and shows fine melanin granules. It contains a large nuclear body in which there is a small chromatin mass. There are now active amœboid movements which do not cease upon exposure to the temperature of the room. The affected blood-corpuscles become enlarged and lose their color. The pigment increases in amount. Toward the end of forty-eight hours the full-grown parasite occupies the greater part of the swollen corpuscle. At this time many of the parasites undergo

the process known as segmentation or sporulation, in which the melanin granules are collected into a compact mass and the protoplasm divides into spores, numbering from 15 to 20, mostly collected into an irregular heap around the pigment mass, sometimes having a radial arrangement. The spores—merozoïtes—which represent the sexually undifferentiated individuals, finally separate from the central mass of pigment granules and from each other, pass into the blood-serum, and, attacking fresh blood-corpuscles, cause subsequent paroxysms of fever. Some of the full-grown tertian parasites do not undergo this process of sporulation, but, attaining a size larger than the red corpuscles, show abundant coarse pigment granules in active commotion and represent the sexually differentiated forms—gametocytes.

Quartan Parasite (*Plasmodium malariæ*).—This species is the cause of quartan fever. Its cycle of development is seventy-two hours. It appears abruptly after the paroxysm in the form of a small, unpigmented body with sluggish amœboid movements on the surface of the red corpuscle. As it increases in size it penetrates within the corpuscle, where it presents an appearance very much like that of the tertian parasite, but smaller. At the end of twenty-four hours melanin granules, coarser than those of the tertian parasite and mostly situated at the periphery, begin to form. As the pigment increases and the parasite develops the amœboid movements become more sluggish and finally cease. Forty-eight hours after the attack the parasites have attained a diameter of one-half to two-thirds that of the corpuscle. In sixty hours they completely fill the corpuscles, of which only a narrow rim of a yellowish-green or brassy tint remains, which in turn presently disappears. The melanin grains assume a radial arrangement and move toward the centre, while the periphery now becomes pigmentless and shows the indication of commencing segmentation, which about the expiration of the third day is complete, each parasite separating into nine to twelve spores, a process corresponding to a fresh attack of fever. Sexually differentiated parasites—gametocytes—persist.

Estivo-autumnal Parasite (*Plasmodium immaculatum; Plasmodium præcox*).—This parasite is the cause of estivo-autumnal fever and the various forms of malarial fever designated tropical, pernicious, and malignant. It is very small, not exceeding when fully developed one-third to one-half the diameter of the red blood-corpuscle. It is very active; its pigment is colored, scanty, and finely granular. The affected corpuscles are frequently shrunken, crenated, and brassy. Its cycle of development is forty-eight hours, but the processes of pigment accumulation and development are not often seen in the peripheral blood since they take place in the spleen, liver, bone-marrow, and cerebral capillaries. The spores are smaller than in the other species, arranged radially, and range in number from 7 to 12. After the illness has lasted some days—never at first—larger, crescentic, ovoid, and round bodies, highly refractive and containing central masses of coarse pigment, are seen. The crescentic and ovoid bodies do not undergo sporulation and represent gametocytes. If the disease continues these bodies increase rapidly and finally may be the only form present.

These sexually differentiated forms—*gametocytes*—are incapable of further development within the human host, but in the abstracted blood

DESCRIPTION OF PLATE XIV.

THE PARASITE OF AESTIVO-AUTUMNAL FEVER. (Drawn by Mr. Brödel for Thayer and Hewetson's paper, The Malarial Fevers of Baltimore, Johns Hopkins Hospital Reports, Vol. V. We copy the original legend.)

1, 2. Small refractive ring-like bodies.

3–6. Larger disc-like and amœboid forms.

7. Ring-like body with a few pigment granules in a brassy, shrunken corpuscle.

8, 9, 10, 12. Similar pigmented bodies.

11. Amœboid body with pigment.

13. Body with a clump of pigment in a corpuscle, showing a retraction of the hæmoglobin-containing substance about the parasite.

14–20. Larger bodies with central pigment clumps or blocks.

21–24. Segmenting bodies from the spleen. Figs. 21–23 represent one body where the entire process of segmentation was observed. The segments, eighteen in number, were accurately counted before separation as in Fig. 23. The sudden separation of the segments, occurring as though some retaining membrane were ruptured, was observed.

25–33. Crescents and ovoid bodies. Figs. 30 and 31 represent one body which was seen to extrude slowly and, later, to withdraw two rounded protrusions.

34, 35. Round bodies.

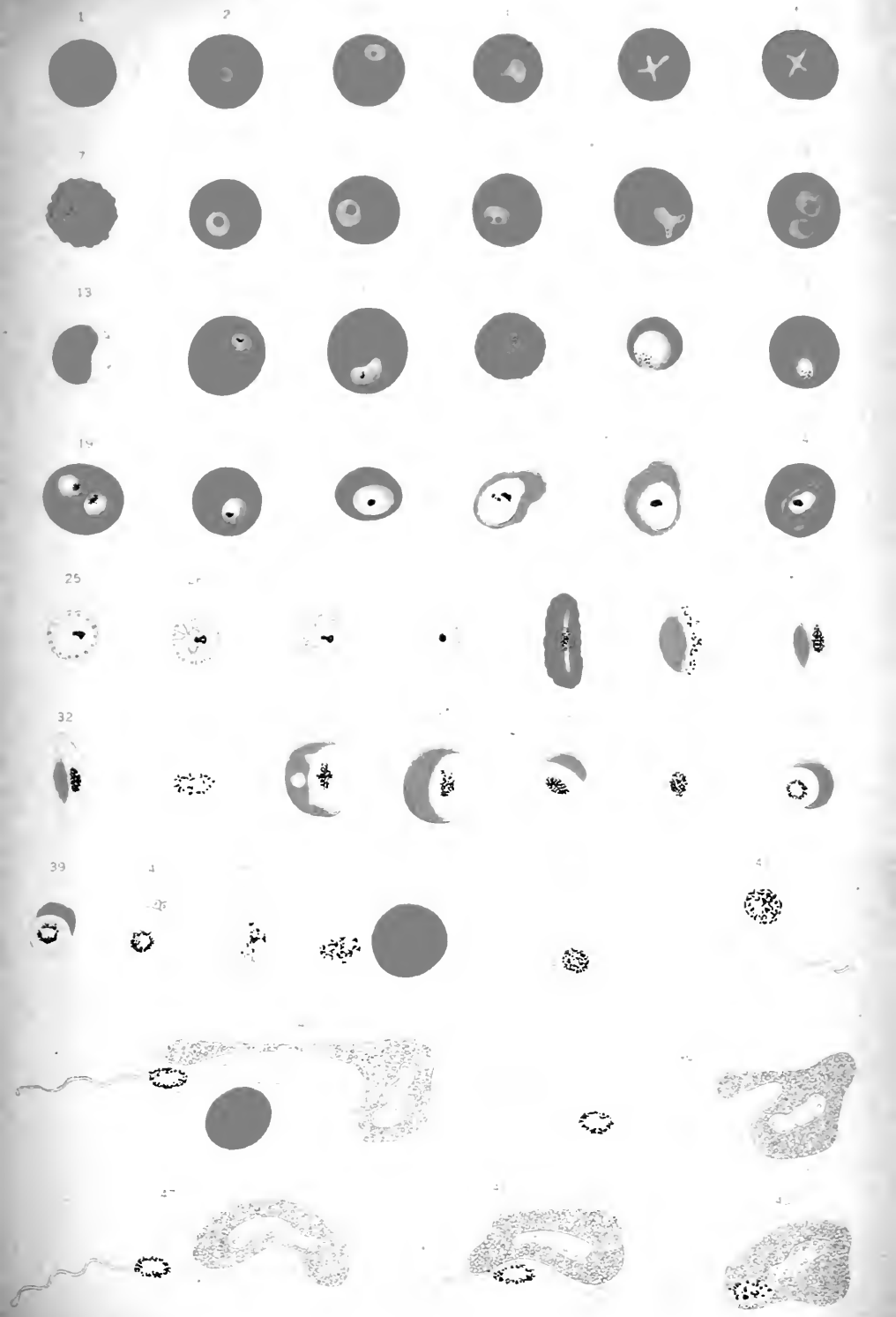
36. "Gemmation," fragmentation.

37. Vacuolization of a crescent.

38–40. Flagellation. The figures represent one organism. The blood was taken from the ear at 4.15 p.m.; at 4.17 the body was as represented in Fig. 38. At 4.27 the flagella appeared; at 4.33 two of the flagella had already broken away from the mother body.

41–49. Phagocytosis. Traced by Dr. Oppenheimer with the camera lucida.

The Parasite of Aestivo Autumnal Fever



upon the slide or in the intestine of Anopheles the male elements—*microgametocytes*—form actively moving flagella—*microgametes*—which detach themselves and penetrate into the coarsely granular female forms—*macrogametes*—which they fecundate.

The independence of the three species of malarial parasites, distinguished by their morphologic and pathologic characters, has been fully established by the results obtained by the experimental inoculation of the blood of patients suffering from malaria into the veins of healthy persons. After a period of latency, varying with the particular species

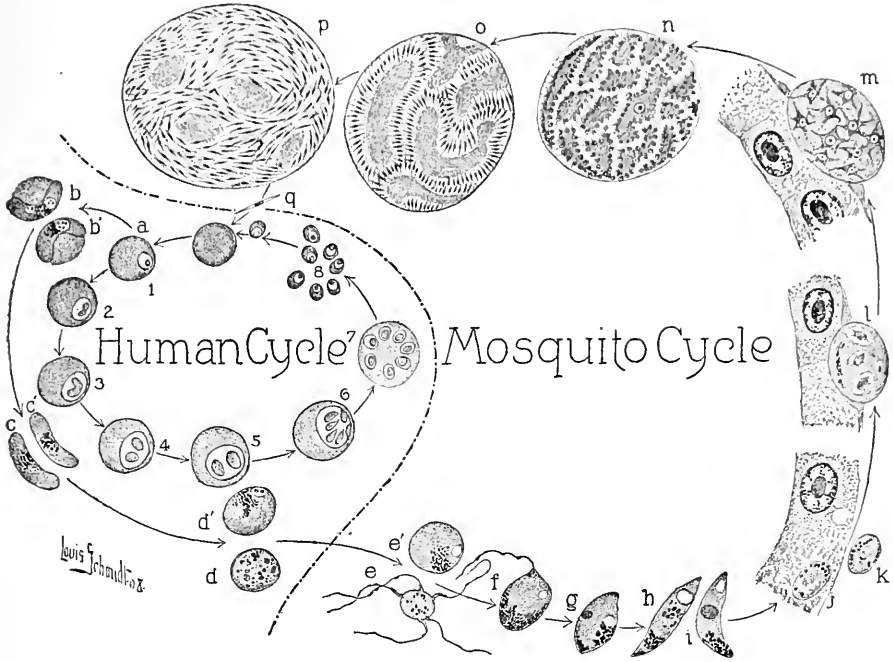


FIG. 286.—Diagram of the complete life-cycle of the estivo-autumnal malaria parasite. 1–7, stages in the development of the asexual cycle (schizogony); 8, spores or merozoïtes; *b b'*, *c c'*, *d d'*, gametocytes in blood of man, sexual cycle; *e'*, macrogamete; *e*, microgametocyte; *f*, macrogamete penetrated by microgamete; *g*, *h*, *i*, motile zygote, vermicule, or oökinete; *j–o*, stages of development in the stomach wall (sporogony); *p*, mature oöcyst showing sporozoïtes; *q*, sporoid.—Based upon the plates of Grassi.

under investigation, the inoculated individuals have developed malarial fever, always of the same type as that of the patient from whom the blood was obtained.

The Parasite in the Mosquito.—The common genera of mosquito in temperate climates are *Culex* and *Anopheles*. Of each there are many species. The Culex appears to play no part in malarial infection, but wherever there is malaria the Anopheles is to be found. If the Anopheles has not been infected by sucking the blood of a human being suffering from malaria, it is of course incapable of communicating the disease by its bite, and the parasite does not develop in cold climates. These two facts serve to explain the presence of Anopheles in regions not malarious. Of the many species of Anopheles described, it is probable that in temperate

climates the A. maculipennis is most active in the diffusion of malaria. The distinction between Culex and Anopheles under ordinary conditions within a house is an easy matter and depends upon the following facts:

Culex: *The Mature Insect.*—The palpi in the female are short, projecting only a little distance beyond the base of the proboscis. The wings show no special markings. Resting upon the wall or ceiling Culex holds its posterior pair of legs turned up over its back, and its body, unless dragged down by the weight of blood, is nearly parallel to the wall or ceiling.

Anopheles.—The palpi in the female are of nearly the same length as the proboscis. The wings of different species show distinct mottlings, hence the names of species—*A. maculopennis, A. punctipennis.* The posterior pair of legs, when the insect is resting, rest upon the wall or ceiling or hang down, and the body is held at an angle of forty-five degrees with the surface upon which it rests. The sex-ripe forms of the malarial parasite, when taken into the stomach of Anopheles, rapidly mature with flagellation and fecundation. The resulting motile fusiform body bores into the wall of the mosquito's stomach and there rests, undergoing a definite cycle of development, with the formation of oöcysts at first oval, later globular. The nuclei within these cysts divide into a great number of daughter nuclei, which form sporoblasts, from which sporozoïtes develop. The mature oöcysts burst and discharge their sporozoïtes into the body of the host. Circulating in the blood these accumulate in the course of a few hours within the cells of the venenosalivary glands and are inoculated with the saliva by the bite of the insect. They are transformed in the blood of the human host into the amœboid form of the parasite and multiply by sporulation (schizogony) until they attain sufficient numbers to produce the paroxysm of fever. The early generations of parasites in the human host are asexual, sexual differentiation occurring later.

The asexual forms serve as the means of prolonging the infection in the human host; the sexual forms, sterile so long as they remain in the human host, become fertile in the mosquito and maintain the life and dissemination of the parasite.

Symptoms.—The cases may be grouped into the regularly intermitting fevers, the irregular, remittent, or continued fevers, and malarial cachexia.

(a) **The Regularly Intermitting Fevers; the Agues.—1. Tertian Fever. 2. Quartan Fever.**—The period of incubation varies from a few days to a fortnight. Latent malarial infection, not attended by symptoms, may be called into activity by removal from a malarious district, other change of climate, or by an attack of illness.

The Paroxysm.—The febrile paroxysm, known as the "chill" or "ague fit," may be divided into three stages: cold, hot, and sweating.

Cold Stage.—There are usually premonitory symptoms, consisting of lassitude, yawning, epigastric distress, sometimes nausea and vomiting, and headache. Shivering occurs and quickly passes into a fully developed rigor with chattering teeth, violent shaking of the whole body, and distressing sensations of cold. The face is cyanotic, the body and limbs covered with goose-flesh, cold to the hand, and showing, when tested by the surface thermometer, a subnormal temperature. At the same time the rectal temperature is high—105°–106° F. (40.5°–41° C.). During this stage

nausea, vomiting, and headache may occur. The pulse is small, frequent, and tense. The duration of the stage varies from ten or fifteen minutes to an hour or more. The danger of the attack lies in the prolongation of the cold stage.

Hot Stage.—The sensations of cold are replaced by those of heat; pallor and cyanosis give way to flushing, and the appearance of collapse is followed by that of more or less intense fever, with bounding pulse, headache, and sometimes delirium. The rectal temperature does not rise, having as a rule reached its maximum about the conclusion of the cold stage. There is urgent thirst and the patient is distressed by subjective sensations of heat. The duration of this stage varies from thirty minutes to three or four hours.

Sweating Stage.—Perspiration starts in drops upon the forehead and face and soon covers the entire body profusely. In some cases the sweating is moderate. The duration of this stage is variable. At its conclusion the patient commonly falls into a sleep from which he awakes refreshed but weak.

The duration of the entire paroxysm varies from an hour or two, as is common among the inhabitants of malarious districts, to six or eight hours. The cold stage is sometimes slight and transient and occasionally not followed by a hot stage. A more common variation from type consists in the hot stage alone, followed by very slight sweating. During the paroxysm the spleen is usually tender and palpable, herpes labialis occurs, and there are the rational symptoms and physical signs of a mild bronchitis, which passes off with the sweating stage.

In the intervals between the paroxysms the patient commonly feels well and regards himself as in his usual health. The paroxysm is the result of a hæmodyscrasia, at once morphological and toxic, produced by the segmentation of the parasites.

1. Tertian Fever.—In this type, caused by the presence in the blood of the tertian parasite, the paroxysms recur every forty-eight hours or every third day. Hence the name tertian. If two groups of parasites, reaching maturity and undergoing segmentation every alternate day, are present, there are daily—quotidian—paroxysms, and the type is double tertian.

2. Quartan Fever.—The paroxysm caused by sporulation of the quartan parasites recurs about the end of seventy-two hours, or every fourth day, and is for this reason known as quartan. If two groups of parasites are present, maturing upon different days, paroxysms occur upon successive days followed by a free day—double quartan; if three groups are present the paroxysms occur daily—triple quartan, likewise quotidian.

The course of the regularly intermittent malarial fevers is greatly influenced by circumstances. Mild cases frequently recover without treatment, especially if removed from the opportunity of further infection and kept in bed. Untreated cases are, however, liable to relapse. The attacks yield promptly to proper treatment by quinine. Repeated reinfection or the persistence of the disease results in anæmia and hæmolytic jaundice, ultimately in malarial cachexia.

(b) **The Irregular, Remittent, Continued, and Pernicious Fevers.**—This type of fever prevails in Southern Italy and Russia, tropical countries, and the Gulf Coast of the United States. Its milder forms occur in temperate climates, chiefly in the latter part of summer and in the autumn, hence the term estivo-autumnal fever. It is associated with the presence in the blood of the parasite of the same name and is characterized by irregularity and intensity. The irregularity is due to the fact that the parasite, which has a cycle of development of apparently forty-eight hours, is subject to great variations in this respect and occurs in multiple groups which do not tend to mature upon certain definite days; the intensity is due to the virulence of the toxins produced by the organisms at the time of sporulation, and their predilection for the cerebral capillaries and perivascular spaces.

The symptoms are most variable. Some of the cases begin as irregular intermittent with prolonged paroxysms, which may occur without chills or chilliness. Another peculiarity is that the temperature rise is gradual and the defervescence by lysis. The tendency to anticipation of the paroxysm is marked, and this feature with prolongation rapidly converts intermittent into a remittent or continued fever. In other cases there is fever of continued type without marked paroxysms, and the clinical picture is suggestive of enteric fever. There is severe headache, flushed face, a bounding but not dicrotic pulse, and enlargement of the spleen. The temperature range is very often 102°–104° F. (38.9°–40° C.) with remissions and exacerbations like those of enteric fever. Intestinal symptoms are not often prominent. The frequent association of a moderate bronchitis with the foregoing symptoms, together with moderate enlargement and tenderness of the spleen, adds to the clinical resemblance. Delirium may occur. It is usually mild but may be active. Subicteroid discoloration of the skin is common and begins early, and in a group of cases—*bilious remittent* of the older writers—deep jaundice is associated with nausea and vomiting and intense headache. The inappropriate and misleading designation, typhomalarial fever, at one time applied to this group of malarial fevers, has fortunately passed into disuse.

The course of the estivo-autumnal fevers is extremely variable. (a) Mild Forms.—In the mildest cases the attack may run its course with moderate fever and indefinite symptoms. The clinical picture suggests simple continued fever or the mildest form of enteric fever—*typhus levissimus*. (b) Severe Forms.—Other cases are more severe. The fever is characterized by marked remissions and exacerbations. There is intense headache, flushed face, delirium, jaundice, and vomiting, with enlargement of the spleen and liver and epigastric tenderness—*bilious remittent fever*. (c) Pernicious Fevers.—The important types are (i) the algid, (ii) the comatose, and (iii) the hemorrhagic.

(i) *Algid Form.*—The attack may begin with a prolonged chill. More commonly there are merely subjective sensations of cold. Gastric symptoms, nausea, vomiting, epigastric distress, are prominent. Extreme prostration occurs and is associated with a feeble, small pulse and rapid, shallow respiration. Frequent diarrhœa, in some instances attended with rice-water discharges like those of cholera, may be present and with it great

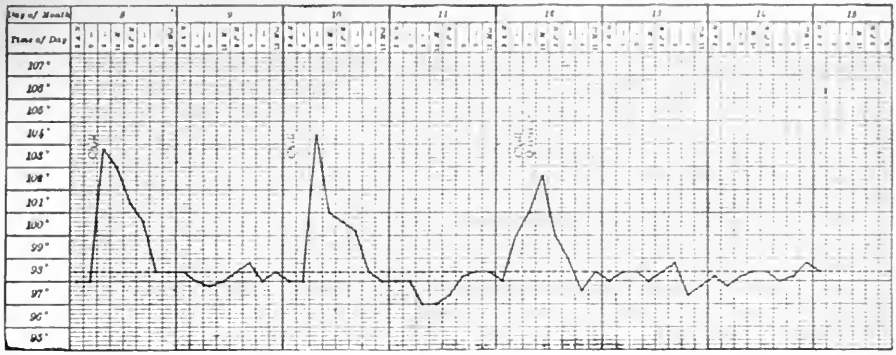


FIG. 287.—Tertian fever.—Craig. *(International Clinics.)*

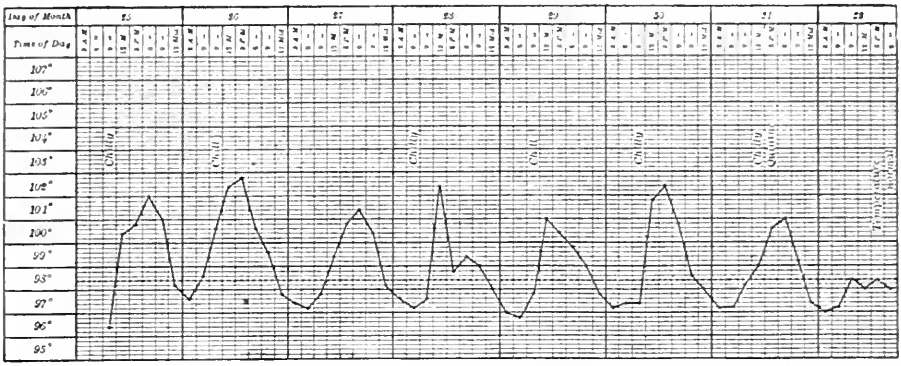


FIG. 288.—Double tertian fever.—Craig. *(International Clinics.)*

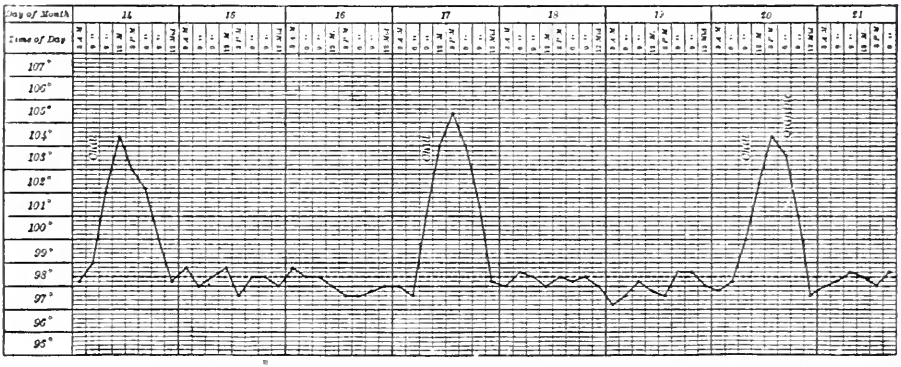


FIG. 289.—Quartan fever.—Craig. *(International Clinics.)*

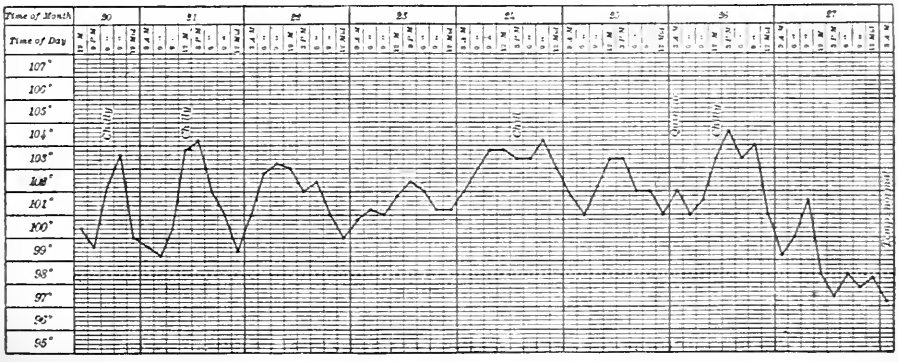


FIG. 290.—Estivo-autumnal fever.—Craig. *(International Clinics.)*

diminution of the urine. Fever is at first absent, the temperature being, as a rule, subnormal. Later irregular febrile exacerbations may occur. In default of energetic treatment death takes place in the course of a few days with the evidences of profound asthenia. Sudoral, syncopal, cardialgic, and choleraic varieties are described.

(ii) *Comatose Form.*—The attack begins abruptly with cerebral symptoms, as intense headache with acute delirium or stupor deepening to coma. In some cases the seizure may be apoplectiform. A chill may mark the onset, but this is not invariably the case. There is a hot, dry skin with high temperature. The patient may die without regaining consciousness; or he may recover consciousness in the course of twelve or twenty-four hours. The second or third attack is usually fatal.

(iii) *Hemorrhagic Form.*—This form is also designated hæmoglobinuria and is identical with the African black-water fever. It is rare in temperate climates, its chief distribution being on the Gulf Coast of the United States, Central America, Lower Italy, and Africa. The disease is malarial, but whether it is due to a special parasite or not remains to be settled. As a rule the patients have suffered from repeated attacks of malarial fever and are in poor health. Parasites have been found in the blood prior to, and in a more limited number of cases at the onset of, the attack. Later they are not found in a majority of the cases. The evidence that malarial hæmoglobinuria is caused by quinine is not conclusive. The attack begins with fever, to which, in the course of a short time, hæmoglobinuria supervenes.

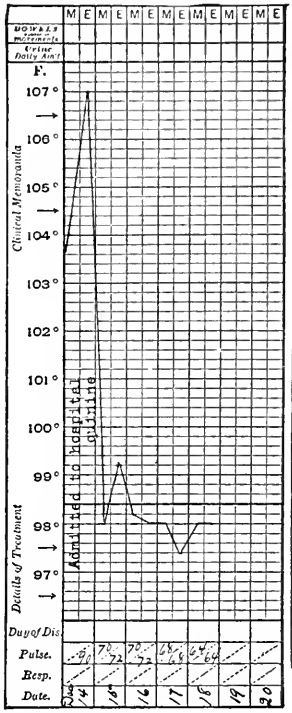


FIG. 291.—Malarial fever. Prompt effect of quinine.—German Hospital.

(c) **Malarial Cachexia.**—Prolonged exposure in a malarious district with repeated infection by way of the parasites is frequently followed by the development of an anæmia of high grade with enlargement of the spleen. Emaciation, a muddy complexion with general cutaneous pigmentation, subcutaneous and retinal hemorrhages, breathlessness upon exertion, œdema of the ankles, are usual symptoms. There is irregular temperature varying from normal to subfebrile ranges with occasional exacerbations—102°–103° F. (30°–39.5° C.). The splenic enlargement is often massive, constituting the tumor known in the Southern States as "*ague cake.*" Hæmatemesis occasionally occurs and may be fatal.

Diagnosis.—DIRECT.—The recognition of the essential nature of the malarial fevers is not usually attended with difficulty. The two important tests are the presence of the blood parasite and the curative effect of quinine. A history of exposure, the well-defined periodicity of the regularly inter-

mitting tertian and quartan fevers, enlargement of the spleen, herpes, and the absence of tuberculosis, syphilis, sepsis, or other causes of periodical fever are diagnostic criteria of secondary importance.

Differential.—The estivo-autumnal form often closely simulates enteric fever. On the one hand the malarial parasite and control by quinine, on the other the Widal reaction and the powerlessness of any drug to arrest the course of the attack, are positive tests. The parasites may be demonstrated in fresh blood taken a few hours before the expected chill. In doubtful cases a carefully prepared cover-slip preparation of the blood may be stained for examination. The intermittent pyrexia which refuses to yield to quinine in daily doses of 15 to 20 grains—gramme 1 to 1.3—properly administered by the mouth or hypodermically is not malarial. The masked intermittents—dumb ague—blurred types—which occur in persistent infections or after inadequate treatment manifest themselves by indefinite symptoms and attacks of irregular fever or an afebrile temperature with an occasional chill. Here the therapeutic test is conclusive. In septic states with intermittent pyrexia, abscess, malignant endocarditis, hepatic fever and the like, a study of the blood is important, since leucocytosis, which does not often occur in uncomplicated malaria, is usually present in these conditions.

Prognosis.—The malarial fevers of the regularly intermitting forms and the estivo-autumnal fevers of temperate climates yield readily to treatment by quinine. If neglected or when reinfection occurs they recur from year to year with vernal or autumnal relapses. Malarial cachexia may thus develop. The prognosis in the tropical forms of estivo-autumnal fever is grave. Life can be saved in many of the cases only by the prompt, skilful, and judicious administration of quinine in sufficient doses, and the removal of the patient from the danger of reinfection. The prognosis in malarial cachexia is fairly good. Health may be regained by proper treatment sufficiently prolonged. The main indication is the avoidance of reinfection.

B. DISEASES DUE TO FLUKES—DISTOMIASIS.

The parasitic trematodes are widely distributed among vertebrate animals. These are important in man: (1) Fasciola hepatica—Distonum hepaticum; (2) Paragonimus westermani—Distoma pulmonis; (3) Fasciolopsis (Distoma) buski; and (4) Schistosomum hæmatobium—Bilharzia hæmatobia. The following clinical forms of distomiasis are to be considered:

1. **Hepatic Distomiasis.**—Several species of liver flukes have been observed in man, of which the most important is Opisthorchis sinensis, widely distributed in the East, especially in Tonquin, China, and Japan. Imported cases have been encountered in the United States. This parasite is 10–14 mm. long by 2.4–3.9 mm. broad. The eggs are oval with a well-defined operculum at the pointed pole. They measure 0.027–0.030 by 0.015–0.0175. The parasite infests the gall-bladder and gall-ducts of domestic dogs and cats and human beings. They have also been found in the pancreas of human beings. Their number is sometimes enormous. The changes produced in the gall-ducts are local dilatations with sacculation and proliferation of the connective tissue of the wall, and in the liver interstitial hepatitis followed by atrophy.

Symptoms.—Intermittent diarrhœa, sometimes bloody; enlargement of the liver, with pain, and jaundice which is intermittent; and slight fever. After two or three years œdema of the feet occurs, followed by anasarca and ascites. The ova are found in the stools. Recovery takes place, but relapses occur. The mortality is about 14 per cent.

2. **Pulmonary Distomiasis.**—Paragonimus (Distoma) westermani has been observed in China, Korea, Formosa, and Japan. Imported cases have been studied in the United States. The body is reddish-brown in color and plump. It is 8–10 mm. in length, 4–6 mm. in breadth. The eggs are oval, brownish-yellow, thin-shelled, and have approximate average diameters of 0.09 mm. in length by 0.06 mm. in breadth. They are found in large numbers in the sputum.

Symptoms.—There is cough and blood spitting but the symptoms are usually slight. The patients are able to follow their occupations. Copious hæmoptysis sometimes occurs. Males are principally affected; women and children rarely. The mode of infection has not been discovered.

3. **Intestinal Distomiasis.**—Fasciolopsis (Distoma) buski has only been observed in the intestine of man. The cases have occurred in Eastern and Southern Asia. Seven cases only have been reported (Braun).

4. **Hæmic Distomiasis—Bilharziasis.**—A parasitic disease endemic in Egypt, Abyssinia, The Sudan, and in many other districts of Africa. There appears to be a centre of infection in Arabia. Elsewhere beyond the borders of Africa it is encountered in imported cases. The parasite—Schistosomum hæmatobium—was discovered by Bilharz in 1852. Unlike the other flukes the sexes are separate and the male carries the female in a gynæcophorous canal. The male is of a whitish color and 12–14 mm. in length, varying from 1 mm. to 0.4–0.5 mm. in diameter. The dorsal surface of the posterior part of the body is covered with spinous papillæ. The female is filiform, pointed at the ends, about 20 mm. in length, and 0.25 mm. in diameter. The eggs are oval, of a transparent yellow color, thin-shelled, and provided with a terminal spine. They vary greatly in size. They hatch in water. The development of the embryo has not been worked out. Whether infection takes place by the mouth, the urethra, or through the skin in bathing is unknown. The young specimens are found in the portal vein, the sexes separate. Hence the males bearing the females penetrate to the venous plexus of the pelvis, from which they reach the wall of the bladder and rectum, the ova being deposited in the tissues but wandering by means of the spine and being voided with the fæces and urine. They are easily found in the latter, especially in the flakes of mucus present. Many remain in the tissues, causing inflammatory irritation, fibroid thickening, and papillomatous growths. Others collecting within the bladder perish and undergo calcification, thus forming the nuclei of the vesical calculi so common in bilharziasis. The ova may be transported to distant parts by the blood stream, and have been found in all the organs, though in small numbers.

Symptoms.—The infection is sometimes latent, the parasites giving rise to no symptoms. This is especially the case while they remain in the portal vein. Early symptoms are catarrh of the bladder, with pain in the

bladder and rectum and in the lumbar region. The urine is at first normal in appearance, but after a time there is tenesmus with bloody mucus and pus at intervals or daily. As the disease progresses the vesical inflammation becomes more intense, the urine contains blood and pus in increasing quantities, and calculi are found which produce their characteristic symptoms. The ureters, the pelvis of the kidneys, the kidneys, the rectum, and occasionally the vagina become involved. The nutrition is greatly impaired and death may result from general marasmus. In a considerable proportion of the cases it is due to uræmia, sepsis, or an intercurrent pneumonia. Perineal and urethral abscess formation is common.

Diagnosis.—The urinary symptoms are suggestive and the direct diagnosis may be made by finding the characteristic ova in the bloody urine or in the blood and mucus discharged from the rectum.

Prognosis.—In mild infections under circumstances in which reinfection can be avoided, the symptoms sometimes disappear. As a rule, the prognosis is highly unfavorable both as to mitigation of suffering and as to recovery.

Katsurada in 1904 described a fluke, closely resembling Schistosomum hæmatobium, to which he attributed an endemic disease, characterized by enlargement of the liver and spleen, cachexia, and ascites, and to which he gave the name S. japonicum. Three months later the same parasite was found by Dr. John Catto and named by Blanchard Schistosoma cattoi. The ova are smaller than those of S. hæmatobium, brownish in color, and not provided with the characteristic terminal spine. This parasite infests the blood-vessels of the intestinal canal and the organs related to it, and the ova are found in the fæces.

In Porto Rico there exists a rectal form of bilharziasis in which the ova are lateral-spined. Sambon has considered this a new species and has called it S. mansoni.

C. DISEASES DUE TO CESTODES.

Tapeworms; Hydatid Disease.

i. Intestinal Cestodes—Tapeworms.

Cestodes are flat worms without mouth or intestine, consisting of a scolex and proglottides, which develop by generation in alternate hosts and by gemmation with elongated tape-like colonies. They combine, except in a limited number of species, the male and the female sexual organs in the same segment. The scolex or head serves as the means of attachment for the entire worm to the wall of the intestine and is for that purpose provided with suctorial organs and clinging organs or hooklets. These organs of attachment are differently arranged in different species. The narrow thread-like part immediately posterior to the scolex is known as the neck. The proglottides or segments are joined to the scolex longitudinally in such a manner that the youngest proglottis is nearest the neck and the oldest most distant from it.

The number of segments varies in different species from a few to several hundred. They are quadrangular and, as a rule, the younger ones have

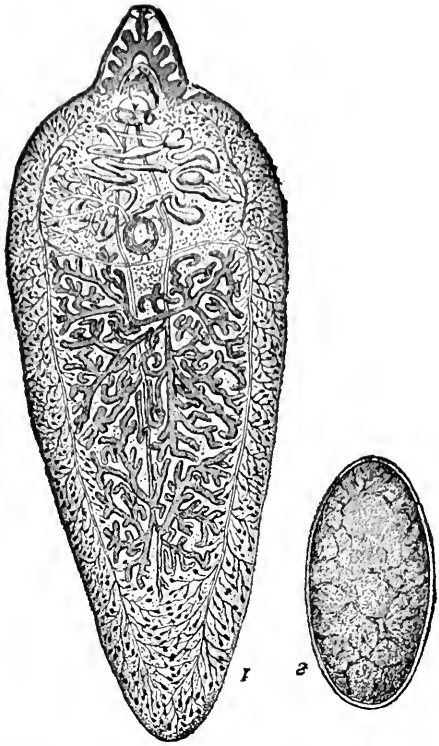


Fig. 292.—1. Fasciola hepatica.—After Claus. 2. Egg of parasite.—After Braun.

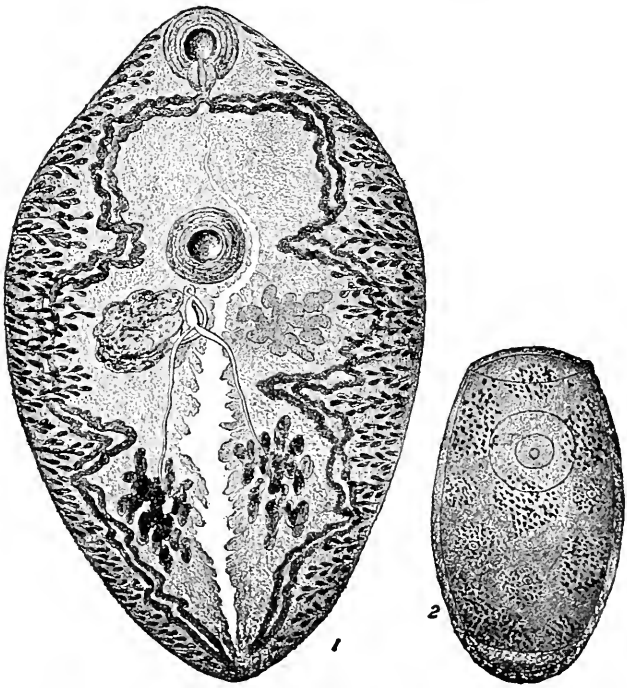


Fig. 293.—1. Paragonimus westermani.—After Leukart. 2. Egg of parasite.—After Katsurada.

their long diameter transverse to the long axis of the worm, those in the middle are squarish, and the most distant have their long diameter in the long axis of the worm. The lateral borders usually converge toward the anterior extremity in such a manner that the anterior border of the segment is shorter than the posterior border of the next younger segment to which it is attached. About the middle of one margin is the projection of the genital pore alternating irregularly. The uterus has a median trunk with lateral branches, which may be seen when the segment is lightly pressed between glass plates.

The segments, single or in tape-like sections of several, become detached from the posterior end and after lingering in the intestine for a time are evacuated with the fæces, or work their way out of the anus and are sometimes found in the clothing of the host. In violent vomiting single or several united segments may be ejected and segments or an entire worm may find the way through abnormal communications into contiguous organs, as the bladder or the peritoneal cavity. The length of tapeworms depends upon the size and number of the segments. The largest species may attain a length of 8 to 10 metres.

The number of genera is about eighty. Certain species in the adult, sexually ripe stage infest the small intestine of man—the definitive host; the corresponding larval forms live normally in the intramuscular connective tissue and viscera of certain animals which constitute the intermediate host. Exceptionally man, by swallowing the embryos—oncospheres—becomes the host of the larval forms—Cysticercus cellulosæ; Echinococcus. The most common tapeworms of man are:

(a) **Tænia Solium** (*Armed Tapeworm; Pork Tapeworm*).—This cestode was so called because it was supposed to exist as a solitary parasite in the intestine. It is now known that two or more tapeworms may be present at the same time.

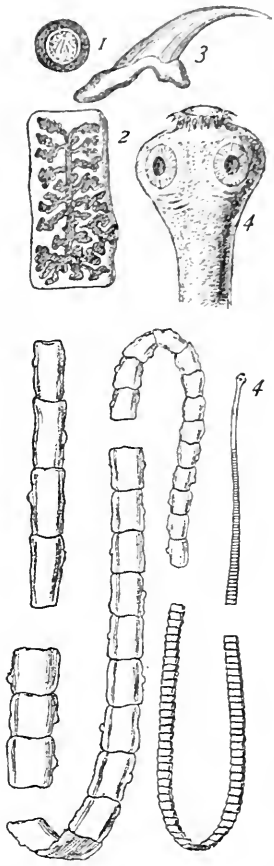


Fig. 294.—Tænia solium. 1, ovum; 2, segment, showing uterus; 3, hook; 4, head.

Average length 3 metres; head globular, 0.8–1.0 mm. in diameter and armed with a double row of hooks; suckers hemispherical; neck slender and 5–10 mm. in length; proglottides 800–1000 in number when mature and ready for detachment, 10–12 mm. in length by 5–6 mm. in breadth; genital pores alternate; uterus consists of a median trunk with 7–10 lateral branches on each side, some of which again branch; eggs oval with very delicate shell; embryonal shell thick, globular, of a pale yellowish color with radial stripes, 0.031–0.036 mm. in diameter; the embryo armed with six hooklets. This parasite when fully developed is found exclusively in the small intestine of man. The embryos are voided with the fæces but undergo no further development unless taken into the stom-

ach of a suitable animal, especially the hog or man himself. The embryo shells are then digested, the armed embryos are set free, and, finding their way to various parts of the body, develop into the larvæ or cysticerci.

The geographical distribution of T. solium corresponds in general with that of the domestic hog and the customary use of raw or insufficiently cooked pork. It is relatively common in North Germany, rare in the United States, and for obvious reasons in Mussulman countries and among the Jews.

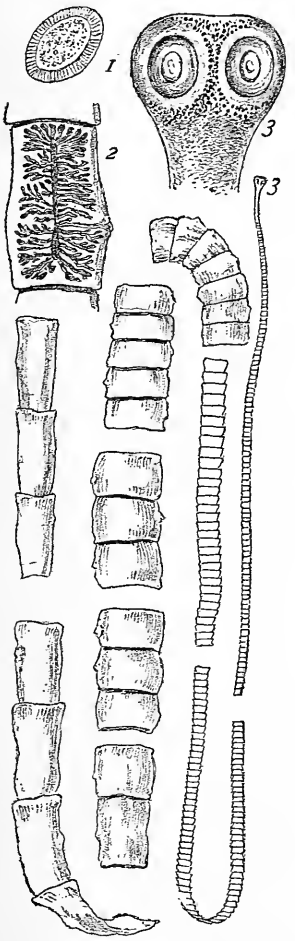


FIG. 295.—Tænia saginata. 1, ovum; 2, fully developed segment showing uterus; 3, head.

(b) **Tænia Saginata** (*T. mediocanellata; Unarmed Tapeworm; Beef Tapeworm*).—Length variable, up to 10 metres, even 36 metres; head cubical, 1.5–2 mm. in diameter and without hooklets; suckers spherical and pigmented; neck long and about half the diameter of the scolex; proglottides average in number 1000; when mature, pumpkin-seed shaped, 16–20 mm. in length by 4–7 mm. in breadth; genital pores irregularly alternate; uterus median with twenty to thirty-five lateral branches on each side, also ramifying. Eggs globular, shell provided with one or two filaments. Embryonal shell oval, thick, transparent, and striated, measuring 0.03–0.04 mm. in length by 0.02–0.03 mm. in breadth. T. saginata in its adult stage is found only in the intestinal canal of man. The ripe segments and ova are voided in the fæces and swallowed by cattle, in the muscles and organs of which they develop into Cysticercus bovis. T. saginata is the most common tapeworm of man and is widely distributed. It is the ordinary tapeworm of North America, is very common in Europe and Africa, and has been known in the East for centuries. The Jews, who are forbidden to eat pork, especially suffer from the beef tapeworm. The eating of uncooked beef is liable to be followed by this form of parasite.

Much less common are:

(c) **Tænia Cucumerina** (*T. elliptica; Dipylidium canium*).—A small tapeworm found in great numbers in the intestines of the dog and cat. The larvæ develop in the lice and fleas of those animals. This parasite is sometimes observed in little children.

(d) **Tænia Nana** (*Hymenolepis nana*).—A small parasite having the genital pores all upon one side. This parasite is common in Italy. Stiles states that H. nana is the most common tapeworm in children in the United States. It is supposed to have cysticercus stages in the intestinal villi, no secondary host being necessary. There are marked nervous symptoms and signs of profound infection.

(e) **Tænia Flavopunctata** (*Hymenolepis diminuta*).—This small cestode is extremely rare. The proglottides show posteriorly a yellow area corresponding to the male sexual organs; hence the name.

(f) **Tænia Lata** (*Dibothriocephalus latus; Bothriocephalus latus*).—The designation of this cestode indicates the lateral pitting of the head and its relatively large size. Length up to 9 or more metres; head almond-shaped, 2–3 mm. in length and flattened, a deep suctorial groove with sharp edges being situated at each side; no hooklets; neck very thin; proglottides numbering 3000–4500, greater in breadth than in length; eggs large with brownish shells, deposited in the intestines, voided with the fæces, and hatched in water. The intermediary hosts are the pike and other fish. This parasite is widely encountered in the Baltic provinces and Switzerland and is the common tapeworm in Japan.

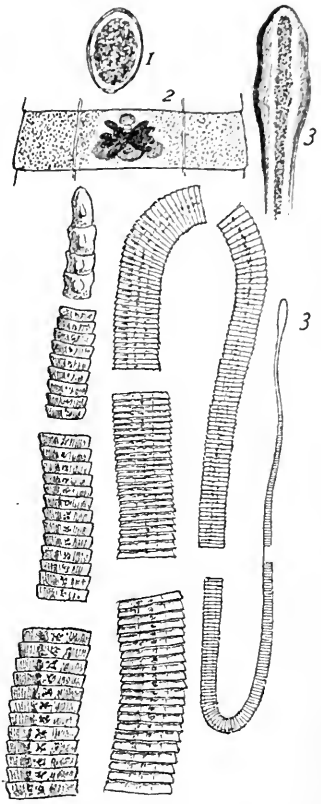


FIG. 296.—Tænia lata. 1, ovum; 2, mature segment; 3, head.

Etiology.—The eating of the raw or insufficiently cooked flesh of animals and fish, and uncleanly habits, constitute predisposing influences of great moment. Tapeworm, owing to systematic food inspection, is rapidly becoming less prevalent in well organized communities. The parasites may be encountered at any period of life. They are common in children and have been met with in infants at the breast.

Symptoms.—Tapeworms may give rise to no inconvenience. They are rarely dangerous. Their presence may, however, occasion symptoms, partly gastro-intestinal, partly nervous. Among the former are a ravenous appetite, abdominal uneasiness and distress, nausea, and diarrhœa; among the latter, nervous depression and hypochondria. Convulsions, chorea, vertiginous attacks, often attributed to the parasite, are rarely directly caused by it. The cessation of any group of symptoms upon its removal is important. Autosuggestion is to be considered. On the other hand troublesome symptoms are sometimes undoubtedly due to intestinal irritation or to toxic substances, evolved by the worm, acting upon the nervous system and the blood—hæmolysis. The Bothriocephalus may be the cause of a severe anæmia having the characters of pernicious anæmia, which sometimes proves fatal, but which in some instances has terminated in prompt recovery after the removal of the worm.

Diagnosis.—The presence of the segments in the stools or in the garments of the patient is positive. The ova and oncospheres may be found in great numbers upon microscopic examination of the stools. Tapeworm treatment should never be inaugurated until the direct diagnosis has been made by the discovery of the segments or ova in the stools. Various sub-

stances found in the fæces, such as shreds of mucus, bits of aponeurosis or tendon, or seeds are brought to the physician by the patient who suspects that he has tapeworm. The differential diagnosis between the ova may be uncertain, but the difference between T. solium and T. saginata is at once apparent upon examining the ripe segments between glass slides. The rare species must be submitted to an expert.

The **prognosis** is favorable. There are several efficient tæniacides.

ii. Visceral Cestodes.

(a) **Cysticercus Cellulosæ.**—Infection of human beings by cysticerci takes place by the introduction of the ripe ova (oncospheres) of T. solium into the stomach. This occurs by drinking contaminated water, eating salads or other raw vegetables washed with such water, or in uncleanly persons by their accidental introduction from the fingers. Autoinfection doubtless frequently takes place in this manner and sometimes from the retropulsion of the mature segments into the stomach in the act of vomiting. The development of Cysticercus cellulosæ takes from two to three or four months. Their length of life in animals is unknown. After a time they die and become calcified or undergo caseation. They have been found in almost every organ in the human body. They appear in subcutaneous tissues and in the muscles as ovoid whitish bodies, on the surface of which a spot may be found which is the invaginated head. They are most common in the brain, in which they sometimes attain considerable size. They infest next in the order of frequency the eye, muscles, heart, the subcutaneous tissue, the lungs, and liver. They were demonstrated by Von Graefe in the vitreous humor and many cases have since been recorded. The number of cysticerci in a single individual varies from a few to several thousand.

Symptoms.—In the hog the cysticerci are often present in enormous numbers without impairing the nutrition or giving rise to noticeable symptoms. In America they are extremely rare in man. When present in small numbers in the subcutaneous tissues or the muscles they cause little or no trouble. When present in large numbers or in regions where their growth is unrestrained by pressure they may cause very marked disturbances. Their general distribution causes muscular pain, stiffness, tingling, and numbness; in the silent region of the brain they may cause no symptoms, but elsewhere they have the same effect as other forms of tumor.

Diagnosis.—Direct.—In the eye a positive diagnosis can be made by ophthalmoscopic examination. Subcutaneous nodules may be excised and examined. The sublingual tissues should be examined in a suspected case. The cysticercus of the ox has been found in man only in a few instances.

(b) **Echinococcus Disease.**—The echinococci are the larvæ of T. echinococcus, a minute cestode measuring 2.5–6 mm. in length and 0.06 mm. in breadth, having a scolex armed with a double row of twenty-eight to fifty hooklets on the rostellum and composed of three or four segments, of which the posterior only is mature. The mature segment contains about 5000 ova. This parasite lives in the small intestine of the domestic dog.

The larval or cysticercus stage is passed in various organs of numerous species of mammals, especially the sheep, ox, and hog. Man occasionally acquires echinococcus by ingesting the oncospheres in caressing or otherwise coming into too close contact with infected dogs, or using the same dishes.

Structure and Development. — Echinococcus or bladder-worm consists of a cyst or vesicle filled with a watery fluid, which may attain in man the size of a child's head but in cattle does not often exceed the dimensions of an orange or apple. The thin wall of the cyst consists of two distinct layers, an external, laminated, cuticular membrane or capsule and

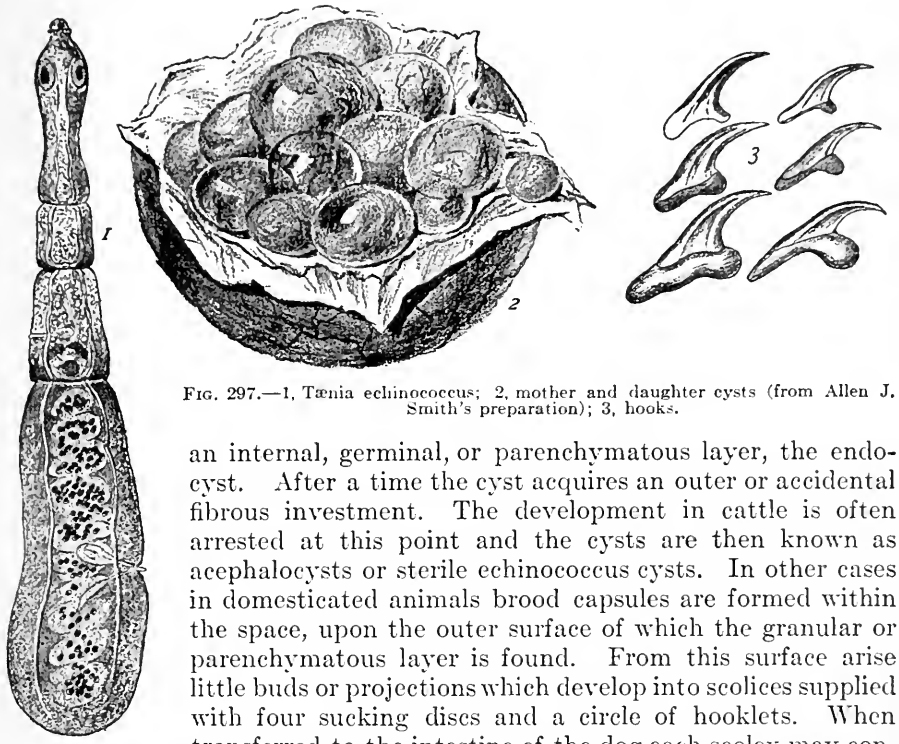


Fig. 297.—1, Tænia echinococcus; 2, mother and daughter cysts (from Allen J. Smith's preparation); 3, hooks.

an internal, germinal, or parenchymatous layer, the endocyst. After a time the cyst acquires an outer or accidental fibrous investment. The development in cattle is often arrested at this point and the cysts are then known as acephalocysts or sterile echinococcus cysts. In other cases in domesticated animals brood capsules are formed within the space, upon the outer surface of which the granular or parenchymatous layer is found. From this surface arise little buds or projections which develop into scolices supplied with four sucking discs and a circle of hooklets. When transferred to the intestine of the dog each scolex may constitute the head of a mature tapeworm. This form is termed Echinococcus veterinorum or fertile echinococcus cyst. In man the mother cyst forms daughter cysts which resemble it in structure and organization and originate from detached portions of the parenchymatous layer. These daughter cysts may develop outwardly and lie between the outer wall of the mother cyst and the adventitious fibrous capsule, or inwardly and, becoming detached from the wall of the mother cyst, float free within the latter. Their number is variable. The daughter cyst may remain sterile or in time may produce brood capsules or granddaughter cysts. Finally the mother cyst may undergo destruction and the daughter cysts, surrounded by thick capsules of connective tissue, may form an irregular tumor mass—multilocular echinococcus. The fluid is of a faint yellowish color, neutral or faintly acid in reaction, non-albuminous, and of a specific gravity of 1.005–1.015.

It contains sugar, inosite, leucin, tyrosin, and succinate of lime and soda. Scolices and hooklets may be found in the fluid of the cysts.

The changes which the cysts undergo are as follows: (a) Death, gradual resorption of the fluid contents, and the conversion of the cyst into a granular, partially calcified mass. Such masses are not uncommon in the liver. (b) Rupture, which may take place into a serous sac, a hollow viscus or a bronchus, the intestine or the bladder, into the bile passages or inferior vena cava, or externally. These accidents are all unfavorable, though recovery may follow the external rupture of the cyst. (c) Suppuration, which may occur with or without rupture and is most frequent in hydatid cysts of the liver.

The geographical distribution of echinococcus disease is wide. In Iceland and Australia it is most common. In European countries it is not rare. It is extremely infrequent in the British Isles and North America.

Symptoms.—The condition is encountered at all ages, but is infrequent in children and old persons. The period of greatest liability comprises the third and fourth decades. Women are more frequently affected than men. The organs most commonly involved are the liver, other abdominal and pelvic organs, brain, and circulatory system. In a majority of the cases one organ only is affected. The primary infection may, however, implicate several organs; later, infection may take place, or in consequence of accidental or surgical traumatism daughter cysts, brood capsules, or scolices may find their way into a serous sac, especially the peritoneum, and colonize, forming new tumors. Hydatids of the liver, when small and deep-seated, cause neither symptoms nor physical signs. When large and superficial they have the attributes of solid or cystic tumors in general. Upon the anterior surface they appear as circumscribed round or oval tumors of firm consistence or obscurely fluctuating; a cyst of the left lobe may displace the heart upward and give rise to extensive dulness in the left hypochondrium; a cyst of the right lobe yields dulness extending upward into the chest. *Hydatid Fremitus.*—When the cyst is superficially situated, it yields in some instances, upon direct finger percussion with the right hand and palpation with the fingers of the left, a peculiar prolonged vibratile tremor. Very large cysts are attended with distressing sensations of weight and dragging, sometimes of actual pain. When suppuration occurs septic symptoms arise. Rupture into the bile passages causes a suppurative cholangitis with deep jaundice; into the vena cava sudden death from the action of the daughter cysts as plugs in arresting the circulation at the tricuspid orifice or in the pulmonary artery. A toxic substance in the fluid contents, probably a leucomaine, causes, when introduced into the peritoneal cavity, a general peritonitis. To this substance has been attributed the urticaria which frequently accompanies the rupture of hydatid cysts or operation upon them.

Diagnosis of Hydatids of the Liver. — DIRECT. — Moderate-sized cysts produce no symptoms by which they can be recognized. A large circular or oval tumor or, in the case of multiple cysts, a similar large, irregular mass, unaccompanied by pain, firm and elastic, or fluctuating, and especially when there is the hydatid tremor connected with the liver and not attended by derangement of the health, justifies a provisional diagnosis of

hydatid cyst. If, upon exploratory puncture, a fluid having the above characters and containing hooklets is withdrawn the diagnosis is positive. If there is a history of acutely developing pulmonary symptoms—rupture into the lung—and hooklets or cysts in the matter coughed up, the diagnosis is certain. The presence of hooklets in a doubtful fluid is diagnostic.

Differential.—*Abscess.*—When suppuration occurs the condition is actually hepatic abscess. The history of a tumor in the hepatic region, unaccompanied by failure of health, is suggestive of hydatid cyst; of dysentery or traumatism in the absence of previous enlargement, in favor of primary abscess. *Syphilis.*—The tumor or tumors are firm and non-fluctuating. The anamnesis is important. *Cancer.*—As a rule the course of the disease is very different from that of carcinoma hepatis; but there are cases in which the multiple tumor formation simulates cancer very closely. Large, single tumors and fluctuation, especially the hydatid fremitus, are in favor of hydatids. *Dilatation of the Gall-bladder.*—Empyema of the gall-bladder, in the absence of adhesions, constitutes a pear-shaped tumor which is often movable in a lateral direction more freely at its lower than its upper extremity. *Hydronephrosis.*—The discrimination is sometimes beset with difficulties. In this condition the tumor may repeatedly disappear with great diuresis. If a hydatid cyst ruptures into the bladder, hooklets may be found in the urine. *Pleural Effusion upon the Right Side.*—The diagnostic difficulties here also are great. Exploratory puncture is necessary. The character of the fluid is distinctive.

Diagnosis of Hydatids of the Lungs and Pleura.—*Lungs.*—The direct diagnosis cannot be made in the case of small cysts which produce only trifling symptoms. Larger cysts compress the pulmonary tissue and lead to inflammation and necrosis with ulceration into bronchi and the discharge of membrane, daughter cysts, and hooklets. Hemorrhage is common. *Pleuræ.*—Hemorrhage into the pleura with empyema and pleuropulmonary fistula occurs. The condition simulates ordinary empyema but the anatomical findings in the sputa—membranes, cysts, or hooklets—are diagnostic. The larvæ may first develop in the pleura and reach a large volume, simulating effusion. The upper line of dulness is irregular. Inflammation may ensue with perforation of the chest wall. The condition is a serious one, liable to be followed by sepsis.

Diagnosis of Echinococcus of the Kidneys.—The kidney may be dilated and simulate hydronephrosis. The diagnosis can be made only by an exploratory puncture and examination of the fluid.

Diagnosis of Echinococcus of the Brain.—The symptoms are not characteristic, being those of tumor—persistent headache with vertigo and vomiting, convulsions of Jacksonian type, and optic neuritis with atrophy. The differential diagnosis cannot be made. Echinococcus cysts in the liver or elsewhere would justify a probable diagnosis. Cystic disease of the choroid plexus is to be considered.

A form of multilocular echinococcus, encountered in men and oxen in Russia, Bavaria, Switzerland, and the Austrian Alps, deserves special consideration. A few imported cases have occurred in North America. The tumor is confined to the liver and consists of dense strands of connective tissue in which are embedded numerous cysts so that the cut section

has a honeycomb appearance. The cysts are filled with a transparent or opaque gelatinous fluid and present the appearance of alveolar colloid cancer. There is a tendency to disintegrating ulceration. The spleen is usually enlarged. Jaundice is common. There is a tendency to hemorrhage and the prognosis is unfavorable.

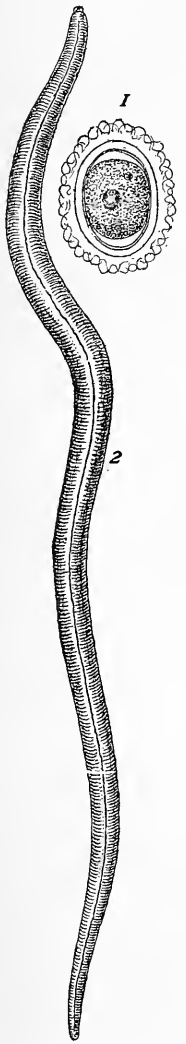


FIG. 298.—Ascaris lumbricoides. 1, ovum (after Braun); 2, female worm.

D. DISEASES DUE TO NEMATODES.

Nematodes are elongated round worms of a filiform or fusiform shape, provided with a mouth and intestinal apparatus. The sexes are in most species separate and the male can be distinguished from the female by its smaller and more slender form and spiral or incurvated posterior extremity. Fertilization takes place within the uterus, and the ova, according to the species, are deposited before or during segmentation or with the embryo fully developed. A few species are viviparous. The mode of infection of the host differs according to the species of worm.

i. Ascariasis.

Ascaris Lumbricoides.—The body is spindle-shaped and of a reddish or grayish-yellow color, with four longitudinal bands and transverse markings. The male measures 12–25 cm. in length and about 3 mm. in transverse diameter at its thickest part; the female from 20–40 cm. by 5 mm. The ova are elliptical with a thick brownish-red covering. They measure 0.05–0.07 mm. in length by 0.04–0.05 mm. in breadth. They are deposited before segmentation and are sometimes present in the stools in great numbers. Generation takes place without intermediate host. This nematode worm is the most common parasite of man and is distributed over the entire world. It is most frequent in young children, but occurs at all periods of life. It is extremely common in the negro races. As a rule only a few worms are present, but cases have been reported in which hundreds of them have been harbored by one individual.

The upper portion of the small intestine is the normal habitat of the round worm. They migrate, however, into the stomach and are frequently evacuated by vomiting. This is especially common in febrile diseases. They sometimes pass through the œsophagus into the pharynx and creep out through mouth or nostrils. They have been known to penetrate the Eustachian tube and appear at the external auditory meatus. They sometimes occupy the biliary and pancreatic ducts, or inflammatory adhesions between the intestine and adjacent parts—*worm abscess*. Passing from the pharynx into the larynx they have caused fatal asphyxia. In other cases they have escaped into the trachea and, penetrating into the

bronchi, have been the occasion of gangrene of the lung. They sometimes find their way into the bladder and are passed with the urine. These wanderings are the cause of most serious and often unaccountable symptoms, but in neurotic persons even the presence of a small number of worms within the intestine may give rise to nervous phenomena—chiefly hysterical—which cease upon their expulsion. In other cases irregular fever with gastro-intestinal symptoms occurs. These symptoms have been regarded as reflex, but are probably due to a toxin. The presence of Ascaris lumbricoides in the intestine may be demonstrated by finding the ova upon microscopical examination of the fæces.

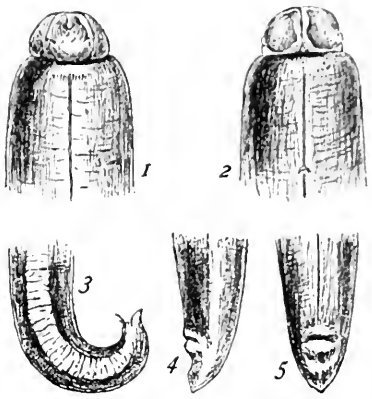


Fig. 299.—1, dorsal aspect of head of Ascaris lumbricoides; 2, ventral aspect of head; 3, tail of male; 4, lateral aspect of tail of female; 5, ventral surface of tail of female—After Claus.

Oxyuris Vermicularis (*Thread Worm; Seat Worm*).—One of the most common and widely distributed of human parasites. The color is whitish and the females may be seen in lively movement in the recently voided fæces of infected persons. The male measures 3–5 mm. in length and the female is 10 mm. in length and 0.6 in breadth. The ova are deposited with the embryo fully developed and are very rarely found in the fæces. This parasite lives in the rectum and colon. It is usually present in enormous numbers. Its wanderings are chiefly nocturnal and give rise to troublesome itching. The localities affected are the sulcus between the nates, the perineum, and the vulva. As the result of scratching, the larvæ may be carried to the nose and mouth and the patient reinfects himself, or he may infect others by the hand directly or indirectly. The primary infection takes place by means of water or fruits or vegetables eaten raw, or perhaps through the intervention of flies. Direct development takes place without an intermediate host.

Oxyuris in rare instances penetrates the wall of the gut and causes perirectal abscess. Most common in children, it may be encountered at any age.

The symptoms in addition to local irritation and itching are restlessness, disturbed sleep, loss of appetite, and anæmia.

ii. Trichiniasis.

The disease is caused by the embryos of Trichinella spiralis, which pass from the intestines and are distributed widely throughout the body, but find the conditions necessary to their further development only in the fibres of the transversely striated muscles, in which they develop into encapsulated larvæ.

Trichinella Spiralis.—The male measures 1.4–1.6 mm. in length and 0.04 mm. in breadth; the female 3–4 mm. in length and 0.06 mm. in diameter. This parasite in the adult stage inhabits the small intestine of man and various mammals, especially the hog. The larvæ are 0.8–1 mm.

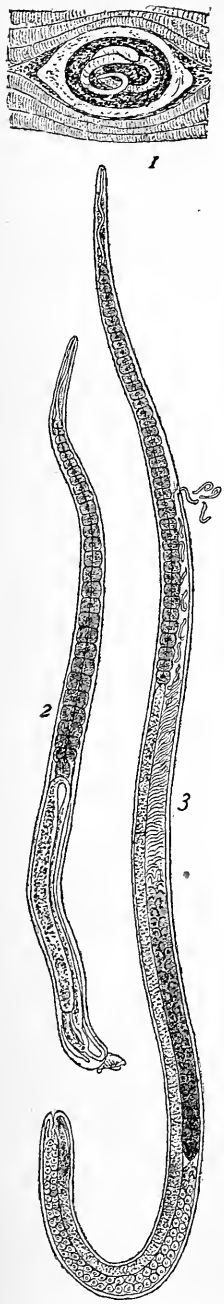


FIG. 300.—Trichinella spiralis. 1, larval worm encapsulated; 2, male; 3, female.

in length and infest the striated muscles, in which they lie coiled spirally in ovoid capsules which mostly have their longitudinal axis parallel with the long axis of the muscular fibres.

It has been experimentally demonstrated that the encysted larvæ, shortly after their introduction into the stomach, are freed from their capsules by the action of the gastric juice and pass into the upper part of the small intestine, where they quickly attain their adult form. Copulation takes place in the course of two or three days, after which the males die and the females, which are viviparous, penetrate the intestinal mucosa and reach the lymph spaces, in which they deposit their young. Carried by the lymph stream and ultimately by the blood current the embryos invade the striated muscles, in which encapsulation takes place. On the ninth or tenth day after ingestion of the affected flesh the first embryos have reached their destination. Two or three embryos may occupy the same capsule. The infested muscular fibres undergo degeneration and lose their striation. The intramuscular connective tissue undergoes an inflammatory hyperplasia and forms the cystic capsule. In the course of several months the capsular walls, at first translucent, undergo calcification, which, beginning at the poles, gradually progresses until in the course of time the enclosed larvæ also become calcified. In hogs calcification is usually long delayed, so that the capsule may elude ordinary examination.

The larvæ are not evenly distributed throughout the muscular system. Favorable locations are the intercostal muscles, the muscles of the diaphragm, abdomen, larynx, and tongue. In their encysted state the larvæ sometimes preserve their capacity for development for many years. The beginning of calcification marks the end of this period. Rats appear to be the normal hosts of T. spiralis. They infect themselves by devouring the flesh of their own kind and the hog waste in abattoirs, and infect other animals, as pigs, dogs, cats, etc., by which they are sometimes eaten. Pigs are also infected by feeding upon the offal of trichinous pigs. Man is infected by eating the raw or insufficiently cooked flesh of infected hogs. The geographical distribution of T. spiralis is much more extensive than the occurrence of trichiniasis in man. The custom of eating raw or only partially cooked pork is the important cause of trichiniasis. Where this custom does not prevail epidemics of trichiniasis do not occur, even though there are great numbers of infected hogs. North Germany espe-

cially suffers and affords many examples of grave epidemics. In South Germany, France, England, and the United States the disease is infrequent. Post-mortem investigations indicate that mild sporadic cases are more common in this country than was formerly supposed and that they are frequently overlooked.

The anatomical lesions consist of minute local wounds of the intestine caused by the boring female trichinellæ and important chiefly in proportion to their number; of the lesions in the muscles, the primitive bundles undergoing granular degeneration with local myositis; and of important changes in the blood, which shows a marked leucocytosis—25,000–30,000. The eosinophiles are enormously increased, comprising 20 per cent. or more of all the leucocytes. Fatty degeneration of the liver and enlargement of the superficial lymph-glands have been described. New broods of embryos are produced from time to time and adult trichinæ are found post mortem in the intestine in cases fatal at the end of four or five weeks.

Symptoms.—Sporadic cases occur, but the disease is more commonly endemic and local epidemics are common. The last can almost always be directly traced to the pork supply and not rarely have followed a "Fest" or entertainment in which uncooked ham, sausages, or similar food have been largely partaken of. Trichinous flesh may be eaten without causing trichiniasis. This occurs when the cysts have been thoroughly acted upon by heat in cooking, when a limited number of embryos have been ingested, and when active purgation has promptly occurred.

(a) Stage of Gastro-intestinal Irritation.—A few days after eating trichinous meat loss of appetite, abdominal pain, vomiting, and diarrhœa occur. These symptoms are of varying intensity, sometimes being absent altogether, sometimes almost choleraic. These symptoms are often attended with great general debility. (b) General Infection.—The invasion of the muscles gives rise in man to a more or less intense myositis, manifested by pain upon movement and pressure, swelling and tension of the muscles, and œdema of the overlying skin. The muscles of mastication and deglutition are especially involved and the predilection of the embryos for the muscles of the diaphragm and the intercostal muscles is the occasion of serious, sometimes fatal dyspnœa. The onset of these symptoms, which follows infection in about ten days or two weeks, is accompanied by fever of remittent or intermittent type—102°–104° F. (39°–40° C.)—and local œdemas, especially under the eyes. Excessive sweating, itching, and urticaria occur. Anæmia, rapid wasting, and loss of strength are common. In the severer cases delirium, tremor, and dry tongue occur. Albuminuria is common and polyuria may occur.

Diagnosis.—Direct.—When a number of persons fall ill at the same time shortly after a festival or who are customers of the same pork butcher, suspicion should be aroused. The finding of the parasites in the pork, in the stools of the patients, in shreds of muscle removed for the purpose under local anæsthesia; muscular tenderness upon movement or pressure; œdema under the eyes; the blood count, showing high leucocytosis with marked eosinophilia, constitute positive diagnostic criteria.

Trichinellæ in the stools, when examined with a low power, appear as short, silvery, glistening threads, which are sometimes still in movement.

Differential.—*Enteric Fever.*—Any resemblance that may occur is superficial. On the one hand we have a definite symptom-group characterized by the gradual rise of temperature, relatively slow pulse as compared with the rise of temperature, palpable spleen, and rose spots, together with a positive agglutination test; on the other the equally characteristic symptom-complex described in the foregoing paragraph. *Rheumatic Fever.*—Pain on movement and tenderness are suggestive. But the joints and not the muscles are involved and the œdema is periarticular. *Cholera.*—The urgency of the intestinal symptoms and the great number of persons simultaneously affected in some of the epidemics has aroused the suspicion of cholera. Rice-water discharges, collapse, and the rapid course of the latter disease are diagnostic points of importance.

Prognosis.—The duration and severity of the attack depends upon the number of the invading embryos. The symptoms are aggravated by the access of fresh groups. In mild cases the symptoms are slight and disappear in the course of two or three weeks. In the more severe cases the active symptoms continue for several weeks and convalescence is tardy. The death-rate varies from 1 or 2 to 20 or 30 per cent. in different outbreaks. Death most commonly occurs in the fourth, fifth, or sixth week.

iii. Uncinariasis.

Ankylostomiasis; Hook-worm Disease.—The parasite of this disease belongs to the Strongylidæ. There are two species parasitic in man, distinguished by specific anatomical differences, especially in the mouth, and by differences in size—the (a) *old-world Ankylostoma duodenale* and (b) *Necator americanus*. The general characters are similar. The body is cylindrical, attenuated anteriorly, and of a reddish color. The males measure 8–10 mm. in length and 0.4–0.5 mm. in breadth; the females 10–18 mm. in length. The eggs are elliptical, thin-shelled, and measure 0.05–0.06 mm. by 0.03–0.04 mm. and are laid in a state of segmentation. In the European species the mouth is supplied with four sharp, hook-like ventral teeth, projecting backward, and with two teeth projecting forward on the dorsal surface, while in the base of the oral cavity there is one tooth directed forward. The cauda bursa of the male has one small dorsal and two large lateral alar processes. The development is direct without an intermediate host.

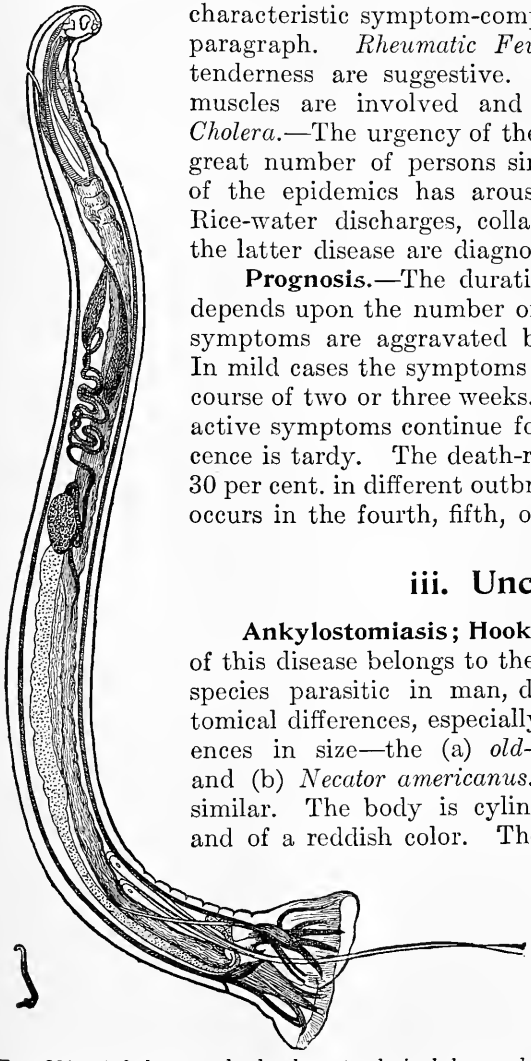


Fig. 301.—Ankylostoma duodenale, natural size below, and much magnified male.—After Schulthess.

The adult worm infests the duodenum, less frequently the jejunum, and sucks blood with its head buried in the mucosa, changing its position from time to time so that minute hemorrhages continue. The number of worms varies from a few to a thousand or more. The duration of life in the bowel is unknown. The disease is essentially chronic. It may be kept up by the prolonged life of the parasite, or by reinfection.

This parasite is the cause of "Egyptian chlorosis," the tunnel disease of St. Gotthard, miners' and brickmakers' disease, and tropical anæmia. It is widely distributed in warm countries, but occurs in all parts of the old world. Since the Spanish-American War uncinariasis has attracted much attention and Necator americanus (Stiles) has been found to be the cause of the so-called southern anæmia. It is endemic in Virginia, North and South Carolina, Georgia, Florida, Alabama, and Texas. It is extremely prevalent in Porto Rico; less so in Cuba and Brazil.

The larvæ live in water and moist soil. There are two hypotheses as to the mode of their introduction: first, that they are ingested by the

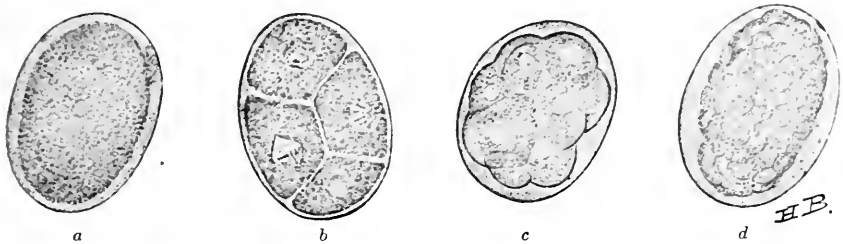


FIG. 302.—Eggs of Uncinaria duodenalis. *a*, unsegmented; *b*, with four segments and showing nuclear spindles; *c* and *d*, later stages of segmentation. × 400.—Emerson.

mouth in drinking water, upon uncooked vegetables, from the soiled hands of men who work and children who play in moist earth, or by clay eaters; and second, that they penetrate the skin by way of the hair-follicles, and are transported by the venous blood to the right side of the heart and the lungs, whence they pass by way of the bronchi and trachea to the pharynx, and are then swallowed. This extraordinary observation of Loos has been confirmed by others, and Smith of Atlanta produced uncinariasis in man by the application of mud containing the larvæ to the arm. The long vexed question of the relation of "ground itch" to uncinariasis is thus settled.

Symptoms.—The clinical phenomena are due to the constant, prolonged drain of blood from the intestinal mucosa by the parasites themselves and from the wounds which they have made, bacterial infection at the site of the lesions, chronic local inflammation and thickening of the bowel, and the deleterious action of toxins produced.

The ova are frequently found in the stools, especially in children, in the absence of symptoms. From this fact it has been inferred that a large number of the parasites are necessary to cause the disease. The anæmia is the most striking condition. Some associated pigmentation gives the skin a peculiar dirty appearance. The facies has been regarded as characteristic, its peculiarities consisting in a pallid, waxy color with faint pigmentation, and a lustreless, blank expression of the eyes. When the disease is marked in children, nutrition and growth are seriously inter-

fered with. Enlargement of the liver and spleen, with œdema, occurs in advanced cases, and the symptoms of anæmia—breathlesnesss and palpitation upon exertion, pallor, puffiness, and headache—are common. The blood shows corpuscular and hæmoglobin reduction, infrequent leucocytosis, and a moderate eosinophilia. In old cases with marked anæmia, which has lasted a long time, the eosinophile count is low.

Diagnosis.—DIRECT.—The presence in fresh fæces of ova showing segmentation, or in older fæces of ova containing the curled embryos within or penetrating the thin shell, is characteristic. The blotting-paper test may be employed. A little of the fæces placed on white blotting paper after an hour will show a reddish color like blood.* Eosinophilia is of diagnostic value.

DIFFERENTIAL.—*Pernicious Anæmia.*—Many of the cases suggest this condition. The presence of the ova in the stools, the locality from which the patient comes, his occupation, the facies, the blood picture, and the therapeutic test with thymol are all to be considered.

Prognosis.—The outlook under thymol treatment, except in advanced cases with marked anæmia, is fairly favorable.

iv. Filariasis.

Filariæ are long, slender nematodes, which live parasitically in the serous cavities and subcutaneous tissues of the mammals which they infest. The males are usually much smaller than the females, having the tail sharply bent or spiral and being supplied with wing-like appendages.

Nineteen genera are described, of which the following are classed under the general term Filaria sanguinis hominis:

1. **Filaria bancrofti.**—The male is colorless and measures 40 mm. in length by 0.1 mm. in breadth. The female is of a brownish color and 70–80 mm. in length, 0.2–0.3 mm. in diameter, and possessed of two uteri which occupy the greater part of the body. The embryos are contained in an elongated, scarcely perceptible membrane in which they move freely. Their length is 0.13–0.3 mm., their breadth 0.007–0.011 mm. By way of the lymph stream they reach the blood and are distributed to all parts of the body. Their appearance in the peripheral circulation is peculiar, showing a remarkable periodicity. During the day there are very few if any to be found; but towards sunset they appear and steadily increase in number until midnight, when they gradually decrease and disappear by the middle of the morning. If the patients sleep during the day and remain awake at night the order of the appearance of embryos is reversed, namely, they are present during the day and not to be found at night. The further development of filariæ is associated with the mosquito, which constitutes the intermediate host.

This parasite is distributed in nearly all tropical countries, and filariasis is common in India, China, Japan, the Australasian Islands, Egypt, The Sudan, Zanzibar, Madagascar, and the Southern United States. Sporadic cases are occasionally encountered in the Middle States and elsewhere in temperate climates in the course of routine examinations of the blood. Some of these cases are imported, but there are instances in which the source of the infection could not be traced.

* Ashford and Igaravidez, U.S. Senate Document No. 808, 1911, mention this test only to condemn it.

Symptoms.—In animals and during a long period of latency in man, filariæ may exist in the blood without causing any inconvenience. Their presence becomes known only upon examination of the blood. After a time anæmia, splenic enlargement, and irregular fever of moderate intensity may occur. When the lymph-vessels become blocked by the adult worm or the ova, characteristic conditions arise, namely, hæmatochyluria, lymph scrotum, and elephantiasis.

Hæmatochyluria.—The urine is opaque, milky white, or blood-tinged. On standing a reddish coagulum sometimes forms. Microscopically there are minute globules and molecular fat and erythrocytes in varying numbers. The quantity of urine is normal or it may be increased. The condition is intermittent, and after passing chylous urine for a short period the patient commonly passes for weeks or even months urine that is normal. The general health is often fair. In other cases there is more or less uneasiness in the lumbar region, anæmia, and vesical irritation, with difficulty in passing the blood-clots which form in the bladder.

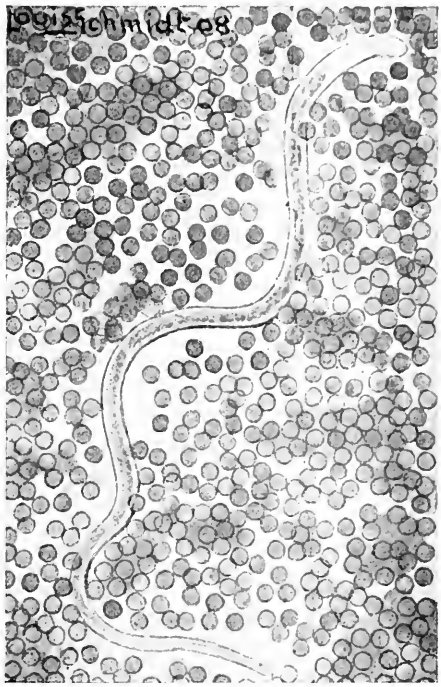


Fig. 303.—Microfilaria nocturna.

Lymph Scrotum.—Blocking of the lymph channels is followed by a dilatation of the lymph plexuses that is sometimes enormous. When the scrotum is involved there is great thickening of the tissues and the distended lymph-vessels are plainly visible. Upon puncture a clear or sometimes a turbid fluid exudes.

A form of elephantiasis follows permanent occlusion of the lymph channels of the lower extremities. The lymphatic glands, especially in the groin, are much enlarged.

Diagnosis.—The living embryos in the recent blood are readily recognized. Their presence is made known by the commotion which they cause among the red corpuscles, and the worm itself, usually in active movement, may be made out with a low power. Chylous urine may occur under other circumstances not well understood. The non-parasitic form is very rare. Withdrawal of fat from the diet is usually followed by disappearance of the chylous appearance, but a glass or two of milk will render the urine again opaque.

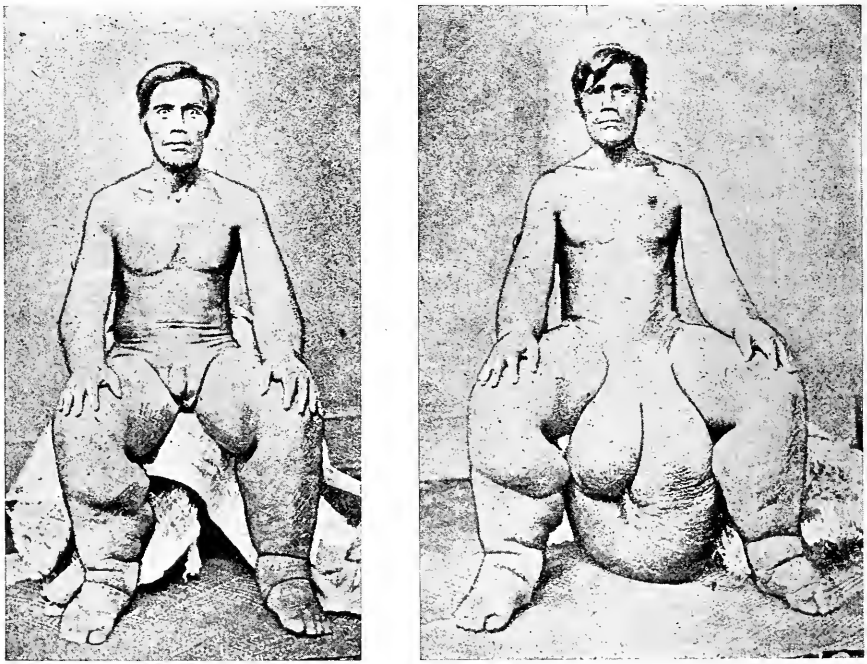
Most cases of elephantiasis in temperate climates are non-parasitic.

Prognosis.—A large proportion of the cases remain latent for an indefinite period. Many of the cases of lymph scrotum and elephantiasis are progressive, and the patient succumbs to exhaustion or intercurrent disease. The removal of an adult worm from the enlarged inguinal lymph-glands

has been followed by the disappearance of the embryos from the blood. If two or more were present this result would not occur.

2. **Filaria diurna.** — This parasite closely resembles F. bancrofti. It appears in the blood during the day only, or at night when the patient remains awake. Manson found the larvæ in the blood of several negroes from Congo. The mangrove fly is supposed to be the intermediate host. F. loa is the adult form.

3. **Filaria perstans.**—The larva was discovered by Manson in 1891. Manson found in the blood of Carib Indians sent from British Guiana two forms of larval filariæ differing in type, one closely resembling those of F.



a *b*

FIG. 304.—*a*. Patient aged twenty-three years, affected with elephantiasis arabum. *b*. Same patient, aged forty-seven years. (*International Clinics.*)

perstans, the other slightly larger and likewise without a sheath. Daniels in 1898 found the adult worm, both male and female. The female is 70–80 mm. in length by 0.12 mm. in breadth; the male 45 mm. by 0.06 mm.; the embryos 0.2 mm. in length by 0.004 in breadth and possess no sheath. The adult worms inhabit the connective tissue of the mesentery at its spinal attachment. This parasite infests the tribes who dwell in dense swamps and deep forests. It abounds on the West Coast of Africa, in British Guiana, and in Porto Rico. It may be observed in the blood both during the night and day. It is thought to be the cause of a pustular disease of the skin common among the negroes of the West Coast of Africa. The intermediary host has not been settled.

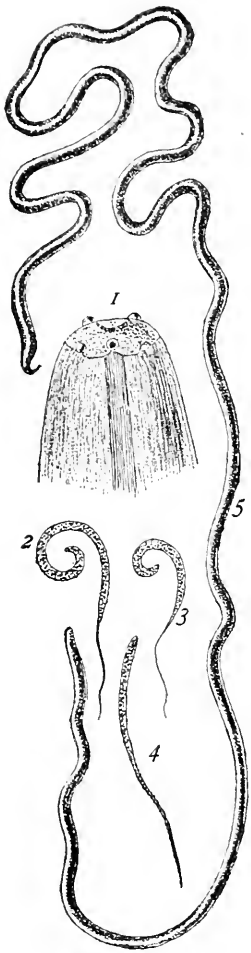


FIG. 305.—Filaria medinensis. 1, head; 2, 3, 4, larvæ; 5, adult worm.—After Claus.

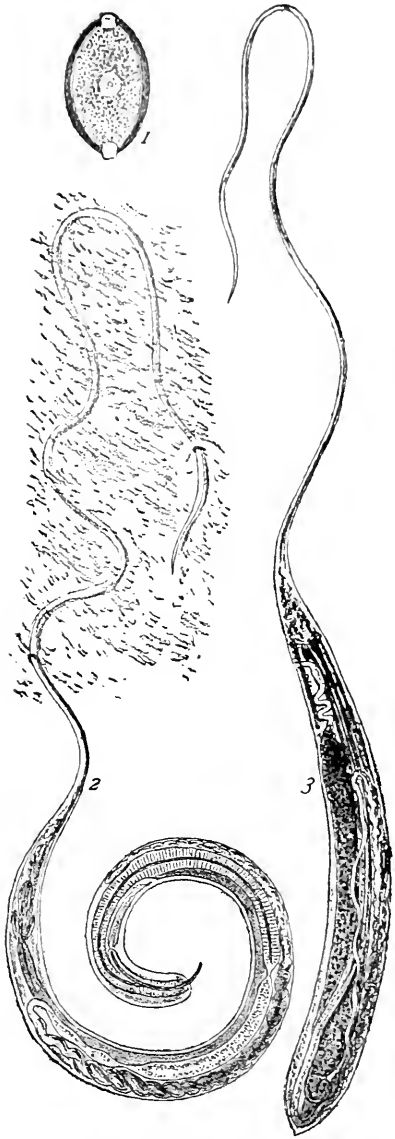


FIG. 306.—Trichocephalus dispar. 1, egg; 2, female; 3, male.

v. Dracontiasis.

Guinea-worm Disease.

Filaria (Dracunculus) medinensis (*Guinea-worm; Medina worm*).—The female measures 50–80 cm. in length and 0.5–2 mm. in diameter and is cylindrical in form with a blunt anterior extremity and a pointed posterior end terminating in a hook. Only the female has been known. Quite recently, however, small worms about 4 cm. in length were found in two instances attached to the females and regarded by Charles, who made the observations, as males. The uterus contains a great number of living embryos, which may reach the open by the rupture of the body of the adult female. They develop in the body of Cyclops. The male and female are probably ingested by the mouth, the former dying and the latter, after impregnation, finding its way to the subcutaneous tissues, in which it slowly develops, remaining quiescent during a period of eight to ten months. It feels like a coil of string under the skin. As the embryos develop the adult worm slowly makes its way downward to the leg or foot, where it forms a small vesicle or abscess, which bursts, leaving an ulcer in the base of which its head appears. The uterus ruptures and the embryos are discharged in a whitish fluid. The worm may now leave the host spontaneously. Guinea-worms are usually solitary, but several have been observed in the same individual. It attacks all races without distinction. It has been known from the earliest historical periods. It is especially prevalent in Africa and the West Indies, but imported cases are occasionally observed in the United States.

When the worm first becomes palpable there is sometimes fever together with an eosinophilia.

Trichocephalus dispar (*T. trichiurus; Whip-worm*).—This parasite may be recognized by the difference between its filiform anterior extremity and its much thicker posterior portion. Its length is 40-50 mm., the male being slightly shorter than the female. The ova are lemon-shaped, and have a thick brownish shell at the ends of which are light yellow plugs or buttons. They measure 0.05 mm. in their long diameter and 0.02 mm. in their transverse diameter. This is a common and widely spread parasite of man infesting the cæcum and other parts of the intestine. It usually gives rise to no symptoms, but exceptionally its presence in great numbers is associated with anæmia and diarrhœa.

A number of less important nematode worms have been observed in man, but the infrequency of their occurrence, and the facts that they do not cause definite internal diseases and that their life history is mostly unknown renders any extended consideration of them at this time inappropriate.

III.

THE DIAGNOSIS OF THE CHRONIC INTOXICATIONS.

I. ALCOHOLIC INTOXICATION; ALCOHOLISM.

The discussion here will be confined to a brief statement of the effects of alcohol on the nervous system. As is well known this poison acts most injuriously upon other tissues of the body also, but these effects are best discussed under other and appropriate headings.

The ravages of alcohol are greater in modern life than among the ancients, for the reason that strong alcoholic drinks are now distilled and marketed at a low price. The first modern writer to attempt to treat this subject adequately was Magnus Huss, who made his observations on the Swedish brandy drinkers.

Pathology.—Changes are found in the coats of the blood-vessels, in the brain membranes, and in the neuroglia, as well as in the kidneys, liver, stomach, and heart. Bevan Lewis and Berkley described with minute care the changes in the central nervous system. According to Lewis these changes are particularly marked in the blood-vessels and neuroglia, and Berkley found microscopic changes in the neurons of the brain. Inflammation of the peripheral nerves, constituting the well-known multiple neuritis, is often seen in chronic alcoholics.

Symptoms.—Alcoholic intoxication is either acute or chronic.

Acute Intoxication requires only a passing notice. In the first stage there is exhilaration, with slight confusion of ideas; then follows a stage in which memory is impaired, self-consciousness is obscured, and the victim may be violent and even maniacal. In the terminal stage a condition of sleep, or even of stupor and coma, supervenes; the pupils are slightly dilated, the face congested or even cyanosed, the breathing normal in frequency, the pulse regular, and the consciousness usually not so lost but that the patient can be roused slightly at least. After some hours he wakes with a sense of depression, headache, nausea, and a dry mouth and throat. Profound intoxication may simulate cerebral hemorrhage, uræmia, diabetic coma, and opium poisoning.

Delirium tremens is caused by protracted acute poisoning. It is the result of a disturbed nutrition of the brain-cells, and continues even after the alcohol is withheld; in fact, it sometimes does not occur until after the patient is deprived of his drink—the *delirium potu suspenso*. This latter form is seen especially in hospital practice—as after a fractured leg, a surgical operation, or even in acute disease, such as pneumonia. The patient may seem to do well for a day or two, when delirium suddenly develops.

Delirium tremens is a psychosis in which the mental faculties are in entire confusion, with terrifying hallucinations, such as the sight of snakes, bats, and other repulsive objects, and the disturbing sounds of voices;

there is restlessness, tremor, incoherence, muttering, insomnia, aversion to food, rapid pulse, and great physical prostration. The so-called typhoid state may develop, with irregular fever and a dry, brown tongue, and the case may end fatally. The prognosis is in the main favorable, except in old, broken-down topers.

Subacute forms of delirium occur, in which the patient is able to be about and to continue his drinking. He is in a dream-like, confused state, irritable, irresponsible, insomnious, unable to attend to his work, eating little, and subject to violent outbursts in which he may even commit murder. Alcoholic *melancholia* is also seen in some hard drinkers, and occasionally leads to suicide. It occurs especially after protracted sprees. The affection known as *mania a potu,* often confused with delirium tremens, is, as its name implies, a state of furious maniacal excitement, in which the inebriate is especially dangerous.

The CHRONIC INTOXICATION from alcohol shows itself in the gradual deterioration, mental, moral, and physical, of the individual. The character and reliability of the man suffer; he is unfit for business or society; subject to moral lapses of various kinds; untruthful, indifferent, cruel, and sometimes dangerous. His memory and his mental faculties generally are impaired. He has a bad color, injected capillaries, a tremor of his hands and tongue, and is a poor eater and sleeper. There is always a chance also that he has a bad liver and bad kidneys. Optic neuritis and atrophy may occur in chronic cases.

Neurasthenia and *hysteria* are among the conditions seen in chronic alcoholics. These complications are seen especially in persons who drink not to such great excess, as steadily—a form of slow chronic poisoning. *Chronic alcoholic insanity* occasionally occurs in confirmed inebriates. The patient has a form of paranoiac deterioration. In addition to the mental failure already noted, he begins to have hallucinations of sight and hearing, somewhat as in delirium tremens. He sees disgusting objects and hears insulting voices, and these are readily excited by every fresh debauch. He then begins to form delusions, often quite well systematized. They are of the persecutory type: he has enemies, who are in league against him, or who would poison him. Sometimes he is markedly hypochondriacal, and has an animal or some unknown disease within him; but his commonest delusion is the delusion of marital infidelity. This is so common as to be held typical by almost universal testimony; and whatever its cause, whether due to failing sexual power or to the natural aversion of the wife, it is in a sense pathognomonic. In time these persecutory delusions may give way to delusions of an expansive type, just as in paranoia, but by that time the chronic inebriate is usually passing into well-marked dementia, and his case is hopeless. Some of these patients under the influence of their delusions commit crime. The terminal stage is one of permanent dementia. In France nearly 14 per cent. of the insane in the asylums are alcoholics.[1] In America the percentage may be not so high.

[1] Semaine Méd., July 10, 1907.

Epilepsy is sometimes a result of alcoholism. It is occasionally seen after prolonged sprees in susceptible individuals, but often it sets in later in life in chronic topers. In the latter case it is usually a sign of beginning degradation of brain tissue. *Alcoholic general paresis,* or pseudoparesis, is a condition which closely simulates the genuine disease. There is the expansive psychosis, with tremor and speech defects, but these patients may recover rapidly when they are deprived of their drink. *Dipsomania* is the overpowering impulse to drink which is seen in a class of excitable and neurotic persons, and which, according to Spitzka, is allied to periodical mania. *Korsakoff's psychosis* is a mental disorder, seen especially in alcoholic multiple neuritis, in which there is mental confusion with loss of identity of time, place, and person, and a tendency to fabulation.

Diagnosis.—The diagnosis of the various forms of alcoholic intoxication is, as a rule, easy. The history in most cases is clear and condition unmistakable.

Mistakes, however, occur in the diagnosis of acute drunkenness, especially in hospital practice when the history is unknown. The odor of the breath is entirely unreliable, for the drunken man, or even the man who has only been drinking moderately, may have opium poisoning, or uræmia, or cerebral hemorrhage, or a fractured skull. In opium poisoning there are contracted pupils and slow breathing, and the coma is more profound, as a rule, than in drunkenness. In cerebral hemorrhage there is usually a hemiplegia, which is shown by the diminished resistance to passive motion on the paralyzed side, and there may be unequal pupils and deviation of the head and eyes. Injury to the head, or fractured skull, is usually detected on careful examination. Uræmia is indicated by the state of the urine and the subnormal temperature, but the latter is seen also in alcoholic intoxication, and the former is not always conclusive. Convulsions are rare in drunkenness, but not unheard of.

Delirium tremens is not likely to be mistaken, but when it breaks out suddenly in a surgical case or in pneumonia it may be confusing. The tremor and peculiar wandering delirium, with frightful hallucinations, are significant. Patients with delirium tremens should always be carefully examined for head injuries, fractured ribs, and other bodily damage; also for pneumonia.

The other forms of alcoholic insanity are usually recognized with ease from the history and the character of the symptoms as given above.

II. OPIUM POISONING; MORPHINISM.

The use of opium and its alkaloid, morphia, has increased greatly in America, until it is now one of the recognized evils of the time. Morphinism is very common among persons whose occupation is attended with the familiar use of drugs as apothecaries, physicians, trained nurses and veterinarians.

It frequently arises in consequence of the medicinal use of narcotics in painful diseases. Psychopathic patients are especially liable to addiction. Husband and wife are very often morphinomanics at the same time. The drug is taken in its crude form, or as laudanum, or occasionally as paregoric, but the commonest practice is to use morphia by the hypodermic syringe. Accidental poisoning is rare, but suicide by laudanum or morphia is more common.

Pathology.—Opium or morphia, when taken habitually, is a slow and insidious poison to the nerve centres, but it does not cause such marked organic changes in the tissues as does alcohol. In old *habitués* there is often a condition of malnutrition or cachexia, shown by a shallow complexion, loss of weight, and gastro-intestinal disorders; but some narcomaniacs show remarkably little physical effect from the drug.

Symptoms.—The effects are acute and chronic. The symptoms of *acute poisoning,* after an initial stage of excitement or dreaminess, are somnolence, passing into stupor and coma, congestion and even cyanosis of the face, full and slow pulse, slow respiration, and strongly contracted pupils. As death approaches the pupils may dilate widely, and they may even be unequal; the respirations are not only slow but also imperfect; the surface pallid, or cyanosed, and covered with a clammy sweat; and the pulse rapid. Trismus and convulsions are rare symptoms. This stage of opium poison closely resembles apoplexy and uræmia, and a positive diagnosis cannot always be made unless the history is clear.

The *chronic effects* of opium poisoning are seen in the habitual users of the drugs, and they are most marked in the nervous system, especially the brain. The ethical sense is perverted. Egotism, habitual mendacity, forgetfulness and irresolution are characteristic. The habitual dose is frequently immediately followed by temporary clearness of intellect and alertness of manner. Abstinence symptoms are often alarming. They consist of restlessness, faintness, nausea and vomiting, sometimes diarrhœa, great anxiety and delirium, all of which are promptly relieved by the habitual hypodermic dose.

Attempts have been made to distinguish the various forms of the opium habit, and their pathological effects. In India opium is eaten; in China it is smoked; and in America it is drunk as laudanum or used as morphia under the skin. To draw distinctions between these varieties of the same vice is, however, hardly possible. Opium, in whatever way it is taken into the system, acts in the end very similarly, especially on the brain and nerves. When taken by the mouth it acts, indeed, more directly and injuriously upon the digestive system; nevertheless old *habitués* acquire a wonderful tolerance. The opium habit becomes a disease, and this disease presents some well-marked symptoms. An abnormal mental state results from the habitual use of the drug. There are moral perversions, enfeebled will, loss of power of attention, and, in advanced cases, a delirious delusional psychosis. In these chronic cases the victim is often insane

and irresponsible, although he may be able to present a fairly good front and to conceal his disorder. There is loss of memory, loss of volition and power of attention, loss of initiative and energy, diminished muscular power, often with trembling, blunting of the higher moral and ethical sense, insomnia, nutritive disturbances, and, finally, illusions, hallucinations, and delusions. The tendency of the morphomaniac to lie and to romance is proverbial. The patient lives so much of his time in an unreal world that he loses his ability to distinguish the true from the false. He becomes both delusional and mendacious. In advanced stages the patient may be violently insane, even maniacal, and may commit acts of violence. Obscure medicolegal problems arise occasionally, for the narcomaniac is sometimes also a kleptomaniac, a pyromaniac, etc.[1]

The physical symptoms vary greatly; in fact, they can hardly be said to be characteristic. Among them are anæmia, rapid exhaustion and lack of endurance, tremor, itching of the skin, anorexia, constipation, wasting, blunting of the special senses, and contracted pupils. The sexual power is weakened and finally destroyed, and in women amenorrhœa is common. Children born of mothers addicted to morphia have a low vitality and often die early.

Diagnosis.—The only rule is to detect the habit. Obscure mental changes, such as those mentioned above, may exist for a long while and not excite suspicion; and he must be a shrewd diagnostician who can detect the morphia taker by his mental symptoms alone. The history, therefore, is all important. Spells of irritability and unrest occur when the victim is deprived of his drug, and are followed by a mysterious calm and serenity when he has taken his secret dose. But such signs require to be interpreted with great caution. Instances are known of judges presiding on the bench, and clergymen preaching in their pulpits, when fully under the influence of their accustomed doses. Diarrhœa, faintness, collapse, are among the graver "abstinence symptoms."

The morphia fiend may also take cocaine, and is occasionally addicted to alcohol, with bromide or chloral as a help. These all-round drug-takers sometimes break down in a state not unlike general paresis—a sort of *pseudoparesis*—from which recovery is possible if they are pulled up in time.

A state of neurasthenia is seen in some morphia takers, especially in women who take small doses and are successful in concealing the habit. Hence the necessity of careful inquiry. The morphia taker often has sores on the skin from the use of the needle.

Heroin has come into general use as a habit drug, especially among high-class defectives and criminals. The employment of Indian hemp for this purpose is less common than formerly. Chloral is occasionally habitually taken by persons of intelligence and refinement, but addiction to this drug is attended with great danger to life. Acetanilide is sometimes employed, but the cyanosis and anæmia which attend its use prevent secrecy.

[1] Wharton and Stillé's Med. Jurisprudence, 5th ed. Chapt. on "Narcomania" by Lloyd, vol. i, p. 874.

III. COCAINE POISONING; COCAINISM.

Erlenmeyer was the first to describe *cocomania* as a distinct disease. The habit has prevailed only in recent years, for cocaine, the active principle of the coca plant, was only discovered by Gardeke in 1855.

Symptoms.—The *physical* symptoms are loss of weight, muscular weakness, tremor, anæsthesia, and disturbance of the heart and circulation.

The *mental* symptoms are marked. No other narcotic makes such a pleasing impression on the brain. It is a sense of well-being, of strength, and of entire happiness. But the duration is brief. Later, as the habit is formed, a delusional insanity occurs, with hallucinations, which may appear suddenly and develop rapidly. The delusions are of the persecutory type, such as fear of enemies, suspicion of marital infidelity, and dread of burglars. The hallucinations are both visual and auditory, and are disturbing or alarming, such as the sight of insects, the sound of insulting words, etc. One of the most characteristic is the so-called "cocaine-bug:" the patient imagines he feels a hard object beneath the skin of his fingers or hand. It is called Magnan's sign, and is probably due to anæsthesia of the sensory nerve-endings.

Cocaine is a virulent poison to the nervous system—much worse and much more rapid in its effects than morphia. Many of these patients, in fact, are also addicted to morphia. Their career is short; they go to pieces quickly and suddenly; and for some time before the final collapse they have their spells of depression, in which there is acute distress. They are also, sometimes, excitable and violent. A peculiarity of some cases is an extreme slowness of the mental processes, and prolixity in speech and letter writing. The habit has much increased lately, especially among the lower classes, even the negroes in some places becoming addicted.

Diagnosis.—This presents no difficulty, as a rule, for the secret will out. It is important to bear in mind that a large number, probably the majority, of cocomaniacs also use morphia. They are worse physical wrecks, as a rule, than the pure morphia takers. Kerr says that the bodily wasting appears quickly; there is great loss of the sense of time; and in fatal cases stupor and coma, with convulsions, occur. Mosso directs attention to what he calls tetanus of the respiratory muscles. In some cases there is great muscular unsteadiness. Insomnia is common, as in all drug takers when deprived of their allowance. While individually these symptoms are common to many conditions, it is the grouping of them that characterizes the chronic poisoning by cocaine. Delusions and hallucinations are rather more common than in chronic opium poisoning. Some of these patients are covered with wounds and scars from the hypodermic needle.

IV. LEAD POISONING; PLUMBISM.

Lead is widely used in the arts; hence lead poisoning in its various forms is not uncommon. The persons most exposed to the poison are the workmen in white lead factories, painters, and smelters. Plumbism occurs also in type-founders, file-makers, shot-makers, gilders, bronzers, lace-makers, glass-cutters, plumbers, and in other artisans.

Chronic lead poisoning is sometimes unrecognized, for the patient may have been exposed in unsuspected ways, as by hair-dye, food stuffs, etc., drinking water, and may not even know himself the nature of his ailment. The chromate of lead has been used by bakers to give color to their products, as reported by D. D. Stewart, of Philadelphia; wines, beer, and cider have been contaminated by lead pipes and lead vessels; and sewing thread is sometimes weighted with lead, so that poisoning has occurred in a seamstress from the constant biting off of the ends.

Pathology.—Lead affects many of the tissues. In the nervous system it causes a peripheral neuritis, and also poisons the brain-cells, but the pathological changes in the brain, according to Berkley, are not as yet clearly demonstrated. Optic neuritis is seen occasionally. The motor cells in the anterior horns of the spinal cord are sometimes affected. Contracted kidneys and arteriosclerosis are not uncommon in chronic lead poisoning. Gastro-intestinal irritation and inflammation are the results of the ingestion of lead. T. Oliver found changes in the liver, such as atrophy of the hepatic cells and increase of connective tissue. Lead is found in the muscles and in other tissues, and may even be detected in the urine and fæces for long periods after its introduction into the system has stopped.

Symptoms.—These are acute and chronic. Acute lead poisoning is marked by pain in the stomach and bowel, vomiting, constipation or diarrhœa, the stools being black from the sulphuret; cramps, neuralgic pains, paralysis, and anæsthesia in the limbs; and finally syncope and collapse. Convulsions and coma occur in some cases. Chronic lead poisoning is known by the following symptoms: a blue line on the gums, anæmia, colic, pain, vomiting, paralysis, anæsthesia, ataxia, tremor, cramps, convulsions, encephalopathy, neurasthenic and hysterical symptoms, optic neuritis or atrophy, contracted kidneys, and the so-called lead gout.

The blue line at the gingival border is very commonly seen, but it has no necessary relationship to the severity of the other symptoms. As a diagnostic sign, however, it is of great value. Anæmia is said to be common among lead miners, and it can occur in any one who is much exposed.

Colic and epigastric and precordial pains are very characteristic. The familiar term "colica pictonum" dates from the seventeenth century when the affection prevailed as an epidemic in Poitou from the contamination of wine with lead. The attacks are often acute, are even seen in persons only recently exposed to lead, and may or may not be associated with wrist-drop. The colic centres about the navel and is often severe and usually without diarrhœa. It may be accompanied with vomiting. This pain and distress about the precordia may simulate angina pectoris or pericarditis. Myalgic and arthralgic pains are also observed, especially about the knees and in the lumbar muscles, and they may simulate gout.

Paralysis is often seen. The commonest form is the familiar *wrist-drop*, caused by involvement especially, but not exclusively, of the interosseous branch of the musculospiral nerve. The supinator longus muscle and the extensor of the metacarpal bone of the thumb, for some unknown

reason, always escape, and as a rule there is no anæsthesia. The paralysis is of the peripheral or flaccid type, with muscular atrophy and reactions of degeneration. The hand falls at the wrist and is almost powerless, for not only are the extensors paralyzed, but the flexors act at such disadvantage that they can only be partially brought into use, as can readily be shown by asking the patient to grasp the observer's hand, when the grip will be found weak; but if the hand is passively extended the grip is much improved. This wrist-drop is always bilateral. In some cases the paralysis is not confined to the extensors of the hand: the muscles of the upper arms are occasionally involved, and the deltoids seem especially prone to suffer. Again, the paralysis may be even more wide-spread, involving the legs, and presenting the form of a more or less generalized peripheral neuritis, with abolished deep reflexes.

A true *progressive muscular atrophy* and a pseudotabes, due to lead, are occasionally seen.

Tremor and cramps are not common in lead poisoning; the former resembles other forms of metallic tremor—it is at first fine, gradually increases in amplitude, and is worse on voluntary movement and during emotion. Anæsthesia likewise is rare in uncomplicated cases. This is true especially of the cases of wrist-drop. Slight hypæsthesia or retardation may occasionally be seen. In the rare pseudotabes various modes of anæsthesia are noted, especially deep anæsthesia, such as alteration in the muscular sense, pressure sense, and sense of position. But there is a form of hysterical anæsthesia which is not so rare, as has been pointed out by the French. It may be of the segmental type and is sometimes influenced by suggestion.

Under the head of *encephalopathy* are included a group of symptoms sometimes seen in lead poisoning. These comprise headache, confusion, delirium, convulsions, and coma. The attack may come on suddenly, and is seen even in persons not long exposed, but it is probably more common in the victims of chronic poisoning. There is sometimes an initial headache, with restlessness and insomnia, or the attack begins abruptly with a fit. The delirium persists for some hours or even days, and is accompanied with hallucinations of sight and hearing. The convulsions may recur, the delirium persisting between them. An isolated attack, without delirium before or after it, may also occur. If the convulsions recur frequently, the patient may pass into an epileptic status, with fever, coma, stertorous breathing, rapid pulse, and failing vitality, and die. If these attacks occur often there is risk of permanent damage to the brain, as is shown by recurring epilepsy, maniacal seizures, melancholia, and dementia. It must not be forgotten that a type of so-called hystero-epilepsy, totally different from the preceding, sometimes occurs in lead poisoning; it is purely hysterical, and is to be known by the hysterical stigmata. Changes in the optic nerves occur in lead poisoning. Atrophy is observed, and it may follow a neuritis. The association of kidney disease with chronic plumbism must not be overlooked, and hence the possibility of albuminuric retinitis.

Neurasthenic and hysterical symptoms may complicate lead poisoning. Among the symptoms are anæsthesia, including hemianæsthesia and segmental anæsthesia, tremor, hysterical paralysis, and hysterical con-

vulsions. The association of gout with plumbism is insisted on by many English physicians. They point to the contracted kidneys, arteriosclerosis, arthralgia, and in some cases arthritic changes and deposits, as evidences of this "lead-gout." Bright's disease is a not uncommon complication of chronic lead poisoning.

Diagnosis.—When the history is clear the diagnosis is easy; but when the history of exposure to lead is wanting, the case may be most obscure. The blue line on the gums is of the greatest value, but it is not always present. The precordial pain in lead poisoning may simulate angina pectoris, but the history and the blue line are usually determinative. The wrist-drop due to lead is always bilateral, although it may be worse on one side; and the supinator longus and the extensor of the metacarpal bone of the thumb escape; thus the case differs from one of trauma of the musculospiral nerve, which is usually unilateral, and involves more muscles. Other forms of paralysis are rare in lead poisoning, but the history and the blue line are usually clear. The pseudotabes due to lead is distinguished from locomotor ataxia by the presence of muscular atrophy and the reactions of degeneration, and the absence of fulgurant pains, Argyl-Robertson pupils, and other true tabetic symptoms. Lead encephalopathy closely simulates uræmia, also idiopathic epilepsy. If the history is obscure the difficulty may be great. The blue line would be important, and there may be an absence of urinary symptoms. If the history is clear there need be no great difficulty in the diagnosis of epilepsy, of uræmia, or of encephalopathy.

The stigmata of hysteria can usually be recognized with a little care. Hemianæsthesia and segmental anæsthesia are not caused by lead alone. Traces of lead in the urine are important.

V. ARSENICAL POISONING.

Chronic poisoning by arsenic is much less common now than some years ago, partly because of more widely disseminated knowledge concerning the dangers of the use of this metal and its salts in the arts and partly because of the enactment of laws limiting the amount of arsenic in wall-papers and dress goods. This form of intoxication is at present occasionally encountered in persons engaged in certain occupations, as the manufacture of wall-papers and other papers, playing cards, book covers, and artificial flowers. Those who handle, and more particularly those who wear, articles of apparel, such as stockings, gloves, and certain dress fabrics, dyed with arsenical pigments in excess, or furs cured by arsenic, are exposed to a theoretical danger which is, however, trifling as compared with that of a generation ago. Not only the greens, as popularly supposed, but dyes of various colors often contain arsenic in dangerous amounts. The danger of acute or chronic poisoning as the result of the introduction of arsenic into articles of food as a preservative is no longer to be considered.

It is a remarkable fact that a large proportion of cases of chronic arsenical poisoning at the present time are caused by the use of this metal for therapeutic purposes. This untoward occurrence may be due to excessive doses, an improper prolongation of the treatment by proper doses,

or to an unusual degree of susceptibility on the part of certain individuals to some of the toxic effects of arsenic. Patients and, in particular, those subject to diseases of the skin frequently continue the use of prescriptions containing arsenic without regularly reporting to the physician. It is important that medical men should protect themselves and their patients by emphasizing the risks attendant upon such a course. The growing use of less toxic arsenical preparations, as atoxyl and sodium cacodylate, will diminish this danger.

Arsenic eating, as practiced in Styria and elsewhere for the purpose of stimulating the powers of endurance and the sexual capacity or improving the complexion, has in many cases resulted in chronic poisoning. The presence of arsenic in beer, derived from the sulphuric acid used in the manufacture of the glucose employed in brewing, recently attracted much attention in England as a source of danger. The use of Paris green and other arsenical preparations as insecticides is attended with little risk in itself. The chief danger lies in having such substances about under conditions in which they may give rise to accidental or intentional poisoning. Miners and smelters of ores containing arsenic are much exposed to the danger of chronic poisoning. Acute arsenical poisoning is rarely accidental, but by no means uncommon in suicidal or homicidal cases.

Pathology.—In the acute form gastro-enteritis, nephritis, and fatty changes in the muscles and viscera, especially the liver, constitute the chief lesions; in the chronic form anæmia, pigmentation of the skin, and lesions of the nervous system, particularly of the peripheral nerves, are common.

Symptoms.—The chief clinical manifestations of chronic arsenical poisoning relate to the skin and the peripheral nervous system. The general health may not be at first greatly impaired. As a rule, however, anæmia, emaciation, loss of strength, cardiac asthenia, and vasomotor derangements are present in varying degree.

The Skin.—Hyperidrosis, glossiness, local ulceration, herpes, and erythromelalgia are occasionally observed. Pigmentation is very common and often marked. It varies from a faint brownish-yellow to a deep brown, and is sometimes distributed over the greater part of the surface; sometimes collected in circumscribed areas and frequently diffuse, with patches of deeper coloring upon exposed parts, in the folds about the joints, and in regions normally pigmented, as the nipples and pudenda, especially in those of dark complexion. Small dense collections of pigment may form and present the appearance of pigmented moles. Patches of pigmentation are occasionally observed upon the mucous membranes. As a rule, to which there are exceptions, the skin, after the exposure to arsenic ceases, gradually resumes its normal appearance. Keratosis is less common. It is usually confined to the palms and soles and occurs in circumscribed patches. Epitheliomatous degeneration may occur. Less common are erythema, which is sometimes symmetrical, polymorphous lesions, furuncles, acne, depraved nutrition of the nails, and alopecia. Puffiness of the eyelids is a common and early symptom.

The Nervous System.—The principal symptoms are those of a peripheral neuritis, which involves the lower extremities much more commonly than the upper, and when both are affected the lower to a greater

degree. Motor derangements, varying from a slight palsy to actual paralysis, may appear in a few hours, or not for several days after a single toxic dose, when the acute gastro-intestinal and depressive symptoms have wholly subsided. In some instances palsy has appeared several weeks after a single poisonous dose, recovery having in the intervening period been apparently complete. In chronic poisoning the paralysis develops gradually after a variable period, the length of which is determined by individual peculiarities and the degree of intoxication. Derangements of sensation consist of paræsthesias of various kinds, and pain. The latter is frequently severe and associated with tenderness along the nerve-trunks. Anæsthesia, hyperæsthesia, and other derangements of sensation occur. The nervous symptoms, both motor and sensory, do not often extend above the knees or elbows, and the thighs and arms, together with the trunk, remain uninvolved. The sphincters escape. In severe cases the muscles of the legs and feet and arms and hands undergo atrophy; the knee-jerks are lost. Reactions of degeneration are present and contractures occur. There are cases in which the resemblance to tabes is very striking. Mental symptoms occur in severe and prolonged cases. They comprise loss of memory, mental weakness, hallucinations of sight and hearing, and may gradually assume the form of a terminal dementia.

Diagnosis.—Direct.—This rests upon the association of the foregoing symptoms, especially those relating to the skin and nervous system, and a history or knowledge of exposure to, or the ingestion of, arsenic. The disappearance of the symptoms upon the removal of arsenic would render the diagnosis positive. When any of the above symptoms arise in a patient under treatment by arsenic for chronic skin disease, anæmia, chorea or habit-spasm, or a person who is employed in arts or manufactures in which arsenic is freely used, or who lives under conditions involving exposure to arsenic, who habitually eats it for any purpose, or who has a history of acute poisoning by arsenic, a provisional diagnosis of chronic arsenical poisoning is justified but should be at once tested by the withdrawal of the patient from exposure to the action of arsenic in any manner whatever.

Differential.—Peripheral neuritis due to other causes, as lead, alcohol, and the infections. Lead palsy may be recognized by the absence of pain, the more common and severe implication of the upper extremities, bilateral wrist-drop without involvement of the supinator longus or the flexors, the blue line on the gums, colic and constipation, and a known history of exposure to lead. Alcoholic neuritis may be recognized by pain, a history of alcoholic excesses, other signs of alcoholism, as the facies, the condition of the mind, or visceral lesions, as for example fatty liver or cirrhosis. Post-infectious neuritis usually shows a clear history of some acute or chronic disease, as enteric fever, diphtheria, or pulmonary tuberculosis. Tabes dorsalis may be distinguished from the rare cases of arsenical poisoning which resemble it, by the gradual onset, the progressive course, the ocular phenomena, the various "crises," a history of syphilis in nearly every case, the lightning pains, and the absence of the cutaneous lesions produced by arsenic. A positive Wassermann reaction and examination of the cerebrospinal fluid are conclusive.

It may be necessary to examine the urine for the presence of arsenic, though it must be borne in mind that arsenic is not always present in the urine when it is known to have been ingested, and that when present it disappears within two or three weeks after its administration has been discontinued. For this purpose it is best for the practitioner to secure the services of a competent chemist.

VI. POISONING BY MERCURY.

As in the case of arsenic, chronic poisoning by mercury is far less common than formerly, because the employment of this metal in the arts is now much restricted and far greater care is exercised in its use in medicine.

Etiology.—As an occupation disease chronic mercurial poisoning occasionally occurs in those employed in making certain dyes, the use of amalgams, the preparation of fulminate, the manufacture of fireworks, among taxidermists, and in those engaged in the making of felts for hats and other purposes (Edsall). Especially hazardous are occupations such as the mining and smelting of mercurial ores. The manufacture of scientific instruments depending upon the physical properties of mercury, as thermometers, barometers, and manometers, involves prolonged exposure to the risk of a slow intoxication. The number of persons engaged in these various occupations is comparatively limited, but for this very reason a knowledge of the effects caused by mercury, while of relatively slight importance to the general practitioner, is of especial interest to the hygienist because they are preventable, and to the diagnostician because they are rare. It is of diagnostic importance that not only those actually engaged in occupations concerned with mercury are liable to this metallic intoxication, but also others who breathe an atmosphere containing volatilized or dust-borne mercury, or drink water in the neighborhood of mines.

Pathology.—Chronic gastro-enteritis, anæmia, emaciation, and fatty degeneration of the viscera are encountered. Neuritis is very rare. Its occurrence has been questioned.

Mode of Access.—Chiefly by inhalation, since mercury volatilizes at ordinary temperatures; to a subsidiary extent through the skin. In medicinal mercurialization most commonly by ingestion; less frequently by inunction. Fortunately ptyalism, mercurial stomatitis (which see), is a danger signal and usually leads to the immediate discontinuance of the drug.

Symptoms.—Ptyalism does not, however, always occur in chronic mercurial poisoning. In a small proportion of the cases it is an early and persistent condition. Early symptoms are headache, disturbed and unrefreshing sleep, and mental and physical depression, especially in the early hours of the day. Anæmia is common. The association of tremor and a curious emotional condition suggestive of hysteria dominate the symptom-complex. Tremor is usually at first absent when the patient is at rest, but shows itself at once upon voluntary movement and is greatly aggravated by emotional excitement. It chiefly involves the hands and lips, but may, in severe cases, affect all the extremities to such an extent as to interfere seriously with the ordinary movements of everyday life. In the early cases the tremor is fine, but later it increases in amplitude and becomes

coarse and rapid. When it affects the muscles of articulation, speech is much deranged and this, with the tremor of the lips and tongue, may suggest disseminated sclerosis. Attacks of vertigo occasionally occur. In grave cases muscular weakness, choreiform movements, and various palsies, together with anæsthesia and other disturbances of sensation, are encountered. These motor and sensory phenomena are, as a rule, irregularly distributed, incomplete, and transitory. Epileptiform convulsions, paroxysmal tonic spasm involving chiefly the flexors of the forearm, and clonic spasms also paroxysmal and affecting all the muscles of the body but without loss of consciousness, have been described. The emotional derangements assume a form highly suggestive of hysteria. But the conclusion that groups of previously healthy individuals collected in one locality from various sources and developing emotional symptoms along with the other phenomena and the tremor, after prolonged exposure to mercurial poisoning, should be hysterical is unwarranted. This group of symptoms includes sensations of weakness, powerlessness, and fright, which, in rare instances, gradually increase to actual obsessions of fear and doubt which render the life of the patient one of abject wretchedness and have in some instances amounted to a form of dementia.

Diagnosis.—Direct.—The fact of habitual exposure to mercury, either in occupation, therapeutically, or under other circumstances mentioned under the subtitle *etiology*, together with the character of the tremor and the emotional disturbances, render a positive diagnosis a comparatively easy matter. If necessary a chemical examination of the urine may be made.

Differential.—The following conditions are to be considered: *Hysteria.*—The presence of the stigmata of this condition (see article on Hysteria) and of the visual phenomena, the intermittent and paroxysmal character of the symptoms in many cases of hysteria, and the absence of the peculiar tremor of mercurial intoxication, are significant. *Disseminated Sclerosis.*—The finer tremor, absence of emotional or hysterical phenomena, the staccato speech, nystagmus, occasional focal phenomena, and the persistent and progressive character of the symptoms, are important in the differential diagnosis. *Paralysis Agitans.*—The passive tremor, unemotional mental condition, peculiar mask-like facies, persistent muscular rigidity and festination of this condition are in strong contrast with the symptom-complex of mercurial intoxication. *Paresis.*—The tremor is suggestive, but the differentiation may be made without difficulty. The emotional states are wholly different. In paresis delusions of grandeur and perversions of the moral sense occur. There are apoplectiform seizures, epileptiform convulsions, remarkable remissions of all symptoms, occasional focal phenomena, and progressive mental and physical deterioration. *Alcoholism.*—The two conditions are frequently associated. The anamnesis, the slight degree of influence of emotional states upon the tremor, and the facies, dilated venules, circulatory and visceral conditions attendant upon chronic alcoholism, are important. *Plumbism.*—The history of exposure to lead, constipation, colic, the gingival line, and the wrist-drop are characteristic.

Prognosis.—In the early cases the outlook is favorable. Even after prolonged and well-characterized symptoms recovery may take place.

In such cases, however, tremor may persist. Persistent palsy, headache, progressive anæmia, and the cachectic state are unfavorable as regards prognosis. Change of occupation and complete freedom from exposure are essential to recovery. If such requirements cannot be met the prognosis is uncertain or unfavorable.

VII. PHOSPHORUS POISONING.

Etiology.—Acute poisoning by phosphorus is rare and usually results from the swallowing of match-heads with suicidal intent. Chronic poisoning is likewise rare and occurs as an industrial disease in localities in which matches are manufactured.

Pathology.—Acute Phosphorus Poisoning.—The coagulability of the blood is reduced; there are diffuse hemorrhages into the skin and viscera and from mucous surfaces; jaundice; fatty degeneration of the muscles and parenchymatous organs, especially of the liver, which also rapidly undergoes enlargement and changes to a bright saffron color. Leucin, tyrosin, cystin, sarcolactic acid, peptones, and sugar are found in the urine and blood. Acid intoxication occurs, as a result of which the urinary ammonia is greatly increased and the urea decreased. In its derangement of metabolic processes phosphorus acts like a ferment.

Chronic Phosphorus Poisoning.—In man the chief pathological change consists of necrosis of the inferior maxilla with suppuration and the formation of sequestra. Exceptionally the upper jaw bones are affected. This process is accompanied by destructive ulceration of the soft parts with more or less abundant pus formation.

Symptoms.—Acute poisoning presents a close clinical resemblance to acute yellow atrophy. Early symptoms are vomiting and diarrhœa, which presently subside, to return in the course of forty-eight or seventy hours, and are then accompanied by intense jaundice, epigastric distress, and diffuse pains in the muscles. At this time petechiæ, submucous hemorrhages, and blood in the vomitus and stools appear. When, as is not rarely the case, match-heads have been taken to produce abortion, this accident frequently occurs. There is profound asthenia, with maniacal excitement which rapidly passes into stupor and coma and is followed by death. The vomited matters may be phosphorescent.

The necrosis of the jaw, which constitutes the chief morbid condition in the chronic form, commonly begins about a single tooth, with caries and abscess formation. The process involves the tooth and surrounding alveolar process and rapidly extends to the neighboring teeth and contiguous structures. The pus is often abundant and very foul. It burrows in various directions and may form sinuses which discharge in the neck. One or several sequestra may form. Rapid anæmia and general sepsis may occur, and in neglected cases amyloid disease, tuberculous infection, and basal meningitis have been observed.

Diagnosis.—Both in acute and chronic phosphorus poisoning, the diagnosis depends largely upon the anamnesis. In the former suicidal intention, or rarely the eating of match-heads by young children; in the latter the exposure to phosphorus, which is volatile at ordinary temper-

atures, as an occupation risk, are of great diagnostic significance. The main points of discrimination from acute yellow atrophy of the liver consist in the period of relief which occurs in the interval between the early gastro-intestinal symptoms in phosphorus poisoning and the graver phenomena, the enlargement of the liver, the occasional absence of leucin and tyrosin or their relatively smaller amount, and the less intense nervous symptoms. There are, however, cases in which, in the absence of a clear history, the differential diagnosis is attended with difficulty. As to chronic poisoning, there are no conditions characterized by similar persistent and extending necrotic processes in the jaw. Actinomycosis may be recognized by the presence of the ray fungus in the pus.

Prognosis.—The mortality in acute poisoning is about 50 per cent. The fatal issue occurs in less than a week. In the chronic form recovery frequently follows early operation. In neglected or very severe cases, even extensive resection may fail to arrest the advance of the necrotic process.

VIII. ACUTE AND CHRONIC POISONING BY ILLUMINATING GAS.

Etiology.—A number of gases enter in varying proportion into the composition of illuminating gas, but carbon monoxide constitutes the chief toxic agent. Water gas is especially active as a poison, because of the relatively large amount of carbon monoxide which it contains.

Acute poisoning commonly occurs as the result of the escape of gas by way of an unlighted burner into sleeping-rooms. This has resulted accidentally from a gust of wind, from leaving the gas turned on, from ignorance, or with suicidal intent. This method of suicide has become very common and when, as is usually the case, the access of air is carefully guarded against and rubber hose, is used it is a very certain one. Acute poisoning may occur, however, in those employed in gas works, and I have seen a case in which it resulted from the escape of gas from a street main. Chronic poisoning is probably rare and is likely to arise among those employed in gas works or those dwelling in houses into which slow but continuous leakage occurs from pipes or fixtures. Toxic symptoms may arise upon breathing an atmosphere containing 0.02 per cent. of carbon monoxide; 0.05 is highly dangerous, and above this the air speedily becomes irrespirable.

Pathology.—The toxic action is chiefly upon the red blood-corpuscles, the oxyhæmoglobin being converted into carbon monoxide hæmoglobin, and the function of the affected corpuscles as carriers of oxygen and carbon dioxide destroyed. The blood is cherry-red in color. There are areas of bluish-red discoloration upon the neck, chest, and elsewhere, visceral hyperæmia, and local hemorrhages. In the chronic cases fatty degeneration of the heart, anæmia, and enlargement of the spleen have been noted.

Symptoms.—The clinical phenomena depend upon the proportion of gas in the atmosphere and the duration of exposure. Progressively they are as follows: malaise, sensations of throbbing, especially in the head, headache, vertigo, muscular weakness, nausea, vomiting, drowsiness, loss

of consciousness, and relaxation of the sphincters. Muscular twitchings and general convulsions occur. In the comatose state there are rapid respiration, a rapid and full pulse, and cyanosis. The blood is cherry-red in color and there is leucocytosis, high in proportion to the gravity of the case. When recovery takes place, sequels relating to the respiratory tract, bronchitis, and bronchopneumonia are common. Lobar pneumonia is rare. Cardiac derangements of a functional kind also occur. To these, in many cases, are added gastric irritability and epigastric pain and tenderness, symptoms of a subacute gastritis. Icterus has been observed and glycosuria is common. Local œdema and various inflammatory and necrotic cutaneous lesions occur. Nervous symptoms are common and important. They comprise various neuralgias and forms of neuritis, tremors and choreiform movements, and neurasthenic manifestations, among which fatigue symptoms are very marked. Amaurosis, nystagmus, and ocular palsies may occur. Persistent headache, dulness of hearing, and tinnitus have been noted. Psychical derangements vary from confusional states with hallucinations to dementia. The foregoing sequels may develop directly after exposure, or they may come on after several days or even some weeks of apparent recovery and undergo gradual intensification. In the suicidal cases the antecedent condition of the patient is to be considered in estimating the actual relation between the carbon monoxide poisoning and the mental state at a more or less remote period after recovery from such poisoning. The symptoms of chronic poisoning are those of the milder forms of acute poisoning continued through an indefinite period, namely, headache, vertigo, nausea, occasional vomiting, muscular weakness, fatigue symptoms, and inability to perform the ordinary physical and mental duties of life. A slow pulse, anæmia, and absence of the deep reflexes have been observed.

Diagnosis.—Direct.—This rests upon the history of the case, which is usually very clear, and the demonstration of carbon monoxide in the blood.

The most satisfactory tests are: (a) Hoppe-Seyler's sodium hydrate test: A solution of specific gravity of 1.30 is added to the blood; if carbon monoxide is present the clot formed is of bright red color, while with normal blood the color of the mass is greenish-brown. (b) Katagama's ammonium sulphide and acetic acid test: To 10 c.c. of blood diluted with water are added, first, 0.2 c.c. of ammonium sulphide solution, and then 0.2 c.c. of 36 per cent. acetic acid. Blood containing carbon monoxide gives a bright red precipitate; normal blood a green precipitate. (c) The Kunkel-Welzer test consists in the addition to the undiluted blood of an equal volume of 20 per cent. potassium ferrocyanide and a small quantity of 36 per cent. acetic acid. Carbon monoxide blood yields a bright red reaction, while the color of normal blood changes to a deep brown. The spectroscopic test may be employed.

In a comatose patient of obscure or uncertain antecedents a positive result may be of great importance, (a) in the immediate diagnosis, (b) in determining the character of later morbid conditions, and (c) from a medicolegal point of view.

Differential.—*Alcoholic Coma.*—The anamnesis is most important. The fact is, however, to be borne in mind that a drunken man may

blow out the gas, turn the burner off and on again, or conclude to end his life by gas poisoning. The history of a spree and the odor of alcohol have, therefore, no positive value in the differentiation of these two conditions. Of greater moment are the appearance of the blood, the above-named tests, cyanosis, and the cutaneous hyperæmia. *Uræmic Coma.*—Urinary suppression, the presence of albumin, and casts, dropsy, and cardiovascular lesions are of importance, particularly when associated with negative results upon testing for carbon monoxide in the blood.

The diagnosis of chronic poisoning by illuminating gas is not readily made. When all the occupants of a dwelling habitually awake with nausea, headache, and vertigo and these symptoms steadily increase, and there is associated muscular and mental weakness and depression, it would become necessary to carefully test the gas-pipes and fixtures. Under such circumstances the gas present in the atmosphere might produce toxic symptoms, though insufficient to give positive reactions either in the air or in the blood of patients. If certain members of the household, upon removal to a different locality, were gradually to recover their health, while those remaining continued ill, chronic gas poisoning would become a sound provisional diagnosis and the point of departure for further systematic investigation.

IV.

THE DIAGNOSIS OF FOOD POISONING.

Certain articles that are always injurious are, sometimes through ignorance or by accident, used as foods. Examples of these are poisonous mushrooms, which contain muscarine, and some species of fish. The poison present in such substances is said to be *endogenous.* Entirely different are the poisons which are occasionally present in foods otherwise normal. These consist of, (a) poisonous metals, (b) animal parasites, (c) fungi, and (d) bacteria. These poisons are *exogenous.*

(a) Poisonous Metals.—This subject is considered under the appropriate headings (see pp. 881, 884, and 887, etc.). The contamination of water in the neighborhood of mines—lead, mercury; of beer by arsenic (outbreak in Manchester in 1900); of wine (lead shot used in washing bottles); the presence of chrome yellow as a coloring matter in cakes; and the possibility that canned vegetables and other foods may become poisonous by slow chemical changes of the lead in the solder or the tin, are to be considered. Under all these conditions the poisoning is chronic, the symptoms are slowly progressive, and a number of persons are affected, very often within the boundaries of a limited district. (b) Animal Parasites.—Certain meats are occasionally rendered poisonous by the presence of trichina or cysticercus (q.v.). (c) Fungi.—The grains used for food may be infected with the ergot fungus, spurred rye—Claviceps purpurea—and become the cause of outbreaks of epidemic disease—*ergotismus.* (d) Bacteria.—Vegetable micro-organisms constitute the chief factor in poisonous foods. Meats obtained from diseased animals may give rise to actual infections.

Foods contaminated with specific infectious organisms, for example, B. typhosus, may cause circumscribed outbreaks of enteric fever. Finally foods infected with saprophytic bacteria, which evolve poisonous products in the substance of the food itself, may become highly poisonous—*ptomaine poisoning*—a term which in popular parlance is used interchangeably with "food poisoning."

I. **Fish Poisoning—Icthyismus; Ichthyotoxismus.**—The toxic substances may be endogenous, as in species of Tetrodon and Diodon, found in Japan and the East Indies. The nature of the poison is unknown. It is located in the testicles and ovaries. In its effects it resembles curare. The symptoms are vertigo, vomiting, dyspnœa, cyanosis, muscular relaxation, and dilatation of the pupils. Death results very rapidly. This is the *fugu* poisoning of Japan. In certain species of sturgeon, pike, and barb a poisonous substance is developed during the spawning season. The intoxication is grave and often fatal, the symptoms being those of an acute gastro-enteritis. Exogenous poisons are more common. If diseased fish is eaten raw, the specific infection may be communicated, or wholesome and edible fish may, if not properly cared for, speedily develop toxic substances associated with putrefactive changes. These substances, in some instances, resist boiling, so that the infected flesh is also injurious after cooking. The intensely poisonous ptomaines are present during the early days of putrefaction, even before changes recognizable by the taste or smell have occurred, while those of a later period are less toxic. The symptoms do not usually appear until after a period of eight to twenty-four hours. There are two groups of cases. In one the clinical phenomena relate chiefly to the nervous system. These are collapse symptoms, with subnormal temperature, abdominal pain, dry mouth, inability to swallow, dull pain in the belly, dyspnœa, and nervous symptoms, as vertigo, dilatation of the pupils, and diplopia. In another group the clinical manifestations are those of an acute gastro-enteritis, uncontrollable vomiting, griping pains, diarrhœa, and profound cardiac asthenia.

Shell-fish.—Poisoning by mussels is not uncommon in Europe and Great Britain. Brieger, in 1885, isolated a ptomaine—*mytilotoxin*—which proved to be highly poisonous. It resists the temperature at which the mussels are cooked, and in this respect mussel poisoning is analogous to poisoning by mushrooms. The poison is not regarded as endogenous but as the result of changes caused by bacteria present in polluted waters. The symptoms are variable, sometimes not occurring until after the lapse of several hours and being choleraic in character, in other cases not showing themselves for a few days and indicating an action of the poison upon the nervous system. They consist of a general urticarious eruption, associated with asthma-like attacks of dyspnœa. Recovery takes place in the course of several days. In other cases the symptoms come on rapidly and resemble those produced by curara. Death has occurred with great rapidity.

Oysters which have begun to decompose, and those obtained from beds in waters defiled by sewage, or laid out in such waters for the purpose of "plumping," frequently cause poisoning, with gastro-intestinal symptoms. Intense and fatal poisoning from this cause is very infrequent.

Much more important is the occasional conveyance of specific infections, especially that of enteric fever, and the causation of epidemics by the eating of oysters and other shell-fish from sewage-polluted water.

Lobsters and crabs, when not fresh, frequently cause symptoms of poisoning similar to those produced by other fish and shell-fish under like circumstances. All kinds of canned fish may, under certain conditions, as decomposition, or infection previous to canning, or injury to the cans, develop poisonous qualities.

II. Meat Poisoning—Sausage Poisoning.—As in the case of fish poisoning, the great majority of the cases are due to bacterial infection, either specific, as in the case of animals infected at the time of slaughter, or accidental, from contact with various articles that are unclean, or from improper care in other respects. In the former case the organisms are those comprised in the *paratyphoid* and *paracolon* groups; in the latter they are saprophytes, as Proteus vulgaris and B. botulinus, or members of the *colon* group. Clinically the cases may be referred to two categories: first, those in which the symptoms chiefly relate to the nervous system, and second, those in which they are gastro-intestinal. Botulismus—allantiasis—sausage poisoning, a specific intoxication caused by B. botulinus, comes on twenty-four or thirty-six hours after eating the food. The symptoms comprise, on the one hand, gastro-intestinal derangements, as epigastric distress, nausea, vomiting, sometimes diarrhœa, sometimes constipation, dryness of the mouth and throat, choking attacks, and stomatitis, with tough, adherent secretion; on the other, nervous disorders, as dimness of vision, mydriasis, diplopia, strangling sensations, aphonia and profound muscular weakness. The pulse and temperature remain normal. Recovery is slow. In fatal cases maniacal delirium, passing into coma, constitutes a terminal event.

III. Poisoning by Milk and Milk Products.—Milk is especially exposed to bacterial infection, and constitutes a favorable culture medium. For these reasons as a raw food it is an abundant cause of intoxications and infections. Boiled or pasteurized milk, properly protected until used, is safe. Pathogenic bacteria may reach milk directly from a diseased animal, as in tuberculosis, or by way of water polluted with excreted matter, as in enteric fever, or from cases of diphtheria or scarlet fever in various ways. The gastro-intestinal diseases of infants in hot weather are due to the bacterial infection of milk. Acute intoxications, in contradistinction to the specific infections, are common, and not only milk itself, but also articles made from it, such as ice-cream, custards, and cream puffs, may give rise to serious poisoning. Vaughan isolated from cheese a poisonous ptomaine, which has been found in milk, but among the milk poisons it is not "the one most frequently present, nor is it the most active one." Of the many different bacteria for which milk forms a culture medium, each has its special toxin. The bacteria which have been especially studied belong to the B. enteritidis group. These organisms do not cause any apparent change in the milk, which presents an alkaline or amphoteric reaction, and is not curdled.

Cheese frequently develops highly poisonous qualities. The accidental introduction of various toxin-producing bacteria and their devel-

opment are very common. The toxins are probably different, but have not yet been fully studied. Tyrotoxicon was the first to be isolated. The symptoms of cheese poisoning are those of acute gastro-intestinal irritation.

Diagnosis.—Food poisoning may be recognized by the history of the case in respect to the eating of certain articles, and the character of the symptoms, which are usually urgent and nervous, or gastro-intestinal, or both. It commonly occurs in circumscribed epidemics, and all the victims are seized at about the same time, which varies according to the nature of the intoxication from a brief period to two or three days. In a suspected case the careful investigation of the antecedent facts, and the character of the symptoms, are sufficient for a provisional diagnosis, which may be confirmed by bacteriological studies, including agglutination tests.

IV. **Grain and Vegetable Poisoning.**—Ergotismus.—The cause is a parasitic fungus—Claviceps purpurea—which grows in the flowers of several grains, especially rye, and is known as ergot. It contains a number of poisons, among which the more important are *sphacelinic acid*, the cause of the trophic or gangrenous form, and *cornutin*, the cause of the nervous or convulsive form of the disease. Ergotismus does not occur in America but is frequently epidemic in certain parts of Europe. It is due to the prolonged ingestion of ergot and is a chronic intoxication, though the onset may be marked by acute symptoms. In the gangrenous form, distant parts of the body, as the toes, fingers, ears, and the tip of the nose, suffer, and the necrosis is preceded by tingling, anæsthesia, spasms, and signs of local congestion. In the nervous form, the chief symptoms are weakness, headache, cramps in the muscles, and contractures. There may be moderate fever with mania, and, in the severer cases, melancholia and dementia occur. There are tabetic symptoms, and at the autopsy sclerosis of the posterior columns has been observed.

Lathyrismus; Lupinosis; Vetch Poisoning.—Chick-pea poisoning occurs in extended outbreaks in Austria, Italy, Northern Africa, and India as the result of the admixture of the powdered seeds of Lathyrus sativus with flour from wheat and other cereals in the making of bread. The symptoms are pain in the lumbar region, girdle sensations, spastic paralysis of the lower extremities, which may increase to complete paraplegia, tremor, and fever.

Pellagra; Maidismus.—An affection probably erroneously attributed to the continued eating of food prepared from fermented or diseased Indian corn. See page 332.

Mushroom Poisoning; Mycetismus.—The symptoms are due to putrefactive and other changes, as autolysis, or the growth of parasitic fungi in the edible varieties; or the accidental ingestion of inedible varieties containing muscarine, phallin, helvellic acid or other toxic substances. They are (1) gastro-intestinal and may come on in the mild cases promptly after the eating of the poisonous fungi, or in the intense or choleraic cases not for several hours; (2) cerebral or nervous and characterized by contraction of the pupils, alternating mania and depression with muscular cramps, salivation, slow pulse and collapse; and (3) hæmatogenous, as subcutaneous œdema and hæmoglobinuria. The last have been produced experimentally by phallin, which has a marked hæmolytic action and causes hæmorrhages

and fatty degeneration of the viscera. On the whole our knowledge of the subject is unsatisfactory. The common edible mushroom is *Agaricus campestris.*

Potatoes.—Local outbreaks of acute poisoning traced to eating potatoes that have sprouted have recently been recorded. The toxic principle is *solanin*—present in considerable amounts as the result of the growth of the Bacterium solaniferum colorabile and B. solaniferum non-colorabile. The symptoms are those of an acute gastro-intestinal catarrh, with headache, jaundice, and great prostration.

Examination of Food in Cases of Suspected Food Poisoning.—As much of the food as can be obtained should be preserved for examination. The quantity is usually small. The investigation should be conducted without unnecessary delay. Meanwhile, perishable articles should be kept on ice without the addition of chemical preservatives. The bacteriological examination should precede the chemical unless there are clear indications of poisoning by definite substances, as arsenic, lead, etc.

The methods comprise animal experimentation by feeding, the injection of sterile water in which the material has been macerated, further injections of such macerations after filtration through a Berkefeld or Pasteur filter, and the determination, when necessary, of the presence of a heat-resisting toxin by injecting the macerations after boiling. Culture and agglutination methods are necessary.

V.

THE DIAGNOSIS OF AUTOINTOXICATIONS.

The term autointoxication is used to designate the intoxications of endogenous metabolic origin. It has been used vaguely for a long time to suggest hypothetical conditions rather than demonstrable facts. Quite recently, under the application of scientific methods, the subject has been to some extent cleared up.

I. **Gastro-intestinal Autointoxication.**—There appears to be no proof that intoxication takes place from the resorption of digestive juices, or of the products of normal digestion, or of the abnormal products of digestion, except in the case of the acetone bodies. Nor has it been demonstrated that, in the normal action of bacteria upon the contents of the alimentary canal, toxic substances are produced. Even in the case of intestinal putrefaction, which, to some extent, is a normal process, an increased amount does not necessarily mean an intoxication. Intestinal putrefaction is largely dependent upon the diet; an excess of protein affords an abundant medium for bacterial growth, yet there is no constant ratio between the protein intake and the output of aromatic substances. An increase of these bodies, especially indican in the urine, actually indicates increased bacterial activity, whereas it is constantly assumed to be the sign of an intoxication. Intestinal putrefaction is to be distinguished from tissue putrefaction, which is the cause of an excess of aromatic bodies in the urine. The aromatic bodies are not in themselves toxic. It has been assumed that other substances of a poisonous character are produced by putrefaction, and that these, like the amount of putrefaction, may be

approximately estimated by the aromatic substances. In point of fact the aromatic substances in the urine afford no indication of the presence or amount of any hypothetical poison and bear no constant relation to the symptoms in any particular case.

Tetany.—Disturbances of the digestive organs—gastric dilatation, pyloric stenosis, carcinoma ventriculi, cholelithiasis, helminthiasis, and gall bladder disease are sometimes associated with mild or severe and chronic forms of tetany. Whether or not poisons produced by the decomposition of food cause tetany has not been determined.

Gastro-intestinal Attacks Associated with Cutaneous Symptoms.—The seizures are acute and recur periodically. They consist of epigastric pain, vomiting, diarrhœa, and various skin eruptions, most commonly urticaria and erythema. Desquamation may occur.

Acute Paroxysmal Gastro-enteritis.—The attack occurs suddenly in the absence of errors in diet, particularly in the absence of food poisoning. The symptoms are vomiting, often uncontrollable; severe abdominal pain; diarrhœa, frequently profuse; and tympanites; together with marked nervous phenomena, as vertigo, spasms, shock, and in grave cases general convulsions and coma.

Intestinal Obstruction.—This condition, either partial or complete, is very commonly attended by symptoms of autointoxication, namely headache, fever, sleeplessness, and albuminuria, with increase in the aromatic substances in the urine; all of which subside when the obstruction is relieved.

Constipation.—Many symptoms are attributed to this condition, but there are few that are constant and none that is characteristic. A furred tongue, poor appetite, headache, lassitude, and mental depression may occur in habitual constipation, but these symptoms are common in those who have a regular daily action of the bowels. There are those who are miserable if the daily morning movement is missed, while others are uncomfortable if by drugs or injections their bowels are moved more frequently than once in the course of some days. *Coprœmia,* a hypothetical intestinal autointoxication from constipation, lacks the support of accurate clinical observation and objective chemical investigation. The work of Horace Fletcher has shown that constipation amounting to the evacuation of small masses of dry fæces at intervals of several days is not incompatible with excellent health. The secondary mechanical effect of the accumulation of fecal matter in the intestines is considered under its appropriate heading.

Gastric Neurasthenic and Other Conditions Vaguely Described as Nervous Dyspepsia.—This group of nervous affections is sometimes attributed, upon wholly insufficient evidence, to autointoxication, and the same statement may be made in regard to a number of nervous diseases, as migraine, neuritis, and epilepsy, and some of the psychoses, as melancholia and forms of dementia.

The Anæmias.—The theory of Sir Andrew Clark in regard to fecal poisoning as the cause of chlorosis rests upon an insufficient basis of fact and is no longer accepted. That pernicious anæmia is probably due to

an autointoxication of intestinal origin finds support in the following facts, namely: that a persistent hæmolysis is the essential pathological process in the disease; that the hæmolytic process is active in the portal system; and that there are, in many of the cases, atrophic changes in the gastro-intestinal mucosa. The nature of the toxic agent has not been demonstrated.

II. **The Retention Intoxications.**—Biliary intoxication is due to the biliary salts and the pigments. The toxic influence is exerted upon the cells of the parenchymatous organs, the muscles, and the blood. Many persons suffer from marked jaundice for considerable periods of time without manifesting evidences of intoxication. Hepatic coma cannot, in the strict sense, be ascribed to cholæmia, since it occurs in cirrhosis of the liver, in which jaundice is a subordinate symptom or absent altogether. It is probably due to derangement of the hepatic functions in metabolism.

III. **Autointoxication from Extensive Abolition of the Function of the Skin.**—Extensive superficial burns are followed by rapidly oncoming collapse, associated with acute degenerative changes in the cells of the parenchymatous organs and muscles, and hæmolysis—evidences of the action of toxic agents, the nature of which is unknown.

IV. **Acidosis.**—Under this term are grouped the derangements of metabolism which result from an excess of acids in catabolism—an acid intoxication. Among the causes of this condition the acids of carbohydrate fermentation in the alimentary canal play an important part.

V. **Gout** in the present state of knowledge may be regarded as an autointoxication dependent upon derangements of the purin metabolism.

VI. *Glycosuria* and *diabetes* may be regarded as autointoxications arising in consequence of faults in the carbohydrate metabolism.

VI.

THE DIAGNOSIS OF HEAT-STROKE AND ELECTRIC STROKE.

HEAT-STROKE.

Heat-stroke is commonly seen in laboring men, and is also not unusual in armies. In the United States Army, from 1868 to 1893, there were not less than 1250 cases, with 47 deaths.

Pathology.—Congestion of the brain and membranes, as well as of the lungs, is common. According to Gihon[1] loss of coagulability of the blood is the one great lesion in *coup de soleil*. Rigor mortis and putrefactive changes occur early. The post-mortem appearances are mostly negative, but there is rigid contraction of the left ventricle of the heart, while the right side and the great vessels contain partly coagulated dark blood. Meningitis is one of the sequels of sun-stroke. The vitochemical changes in the blood, muscles, and nerve-centres are not fully understood.

Symptoms.—Two forms are recognized: simple heat exhaustion, and heat-stroke proper.

In *heat exhaustion* the patient usually collapses, and may even fall in a partial or complete syncope. The surface of the skin is cool, the pulse rapid and feeble, and the temperature may even be subnormal—as low as 95° or 96°. In the worst cases there is sometimes mental confusion, and delirium has been occasionally reported. The prognosis in these cases is usually good, if the patient's general health is sound.

In *heat-stroke proper* the chief symptoms are as follows: headache, oppression in the epigastrium, sometimes nausea and vomiting, a sense of weakness, vertigo, dimness of vision, and unconsciousness, with fever and rapid pulse. Coplin, among the sugar refiners of Philadelphia, also describes a "cramp" in the epigastrium, and sometimes in the back and the calves of the legs, as among the premonitory symptoms. Of the various symptoms the only one that can be called pathognomonic is the exceedingly high temperature. Richards, in the Rhode Island Hospital, observed temperatures ranging as high as 110°, and Packard, in 31 cases in the Pennsylvania Hospital, saw the temperatures range up to 110°, 111°, and even 112°. These are extreme cases, usually with contracted pupils and profound unconsciousness, and many of these patients die. Death sometimes occurs so quickly that a special or *apoplectic* type is recognized, and if dyspnœa is prominent, the type is called *asphyxial*. Most authors are in accord about the contracted pupils, but an exceptional case of dilated pupils has been noted, and as death approaches the pupils may dilate. Convulsions are not common. Great oppression of breathing is sometimes experienced, with a sense of constriction of the chest. Pirrie, in his cases in Central India, observed priapism and seminal emission just before the seizure. Alcoholism is an active promoter of heat-stroke. Most cases occur in persons who have been over-exerting themselves.

[1] "Heat-stroke," in XX. Cent. Pract., vol. iii., p. 253. This article by Gihon is a useful review of the whole subject, both historical and clinical.

Diagnosis.—Heat-stroke must be distinguished from cerebral hemorrhage, uræmia, alcoholic intoxication, and opium poisoning; but from all these conditions it differs in its history and its high temperature. The history alone is so clear and suggestive in most cases that a mistake is hardly possible. In uræmia a subnormal temperature is common, and the condition of the urine is characteristic; if the temperature rises, as it does in some cases, especially towards the end, it does not mount as high as in sun-stroke. The contracted pupils in heat-stroke may suggest opium poisoning, but in the latter there is slow respiration, and in the former high temperature. In mere alcoholic intoxication we do not see pyrexia, much less hyperpyrexia, nor contracted and immobile pupils. In fact, in all comatose conditions, as in those just named, and in diabetic coma, we do not see high fever, nor is there the history of exposure to heat. Injury to the head can usually be excluded by the history and by careful physical examination. In cerebral hemorrhage there is usually hemiplegia, which can be recognized as a rule by the difference in resistance on the two sides. The temperature often rises as death approaches. The attempt to distinguish sun-stroke from heat-stroke is not called for.

ELECTRIC STROKE.

Under this heading are included both lightning stroke and shocks from dynamos. The vast extension of the use of electricity in recent years has made these accidents not uncommon, and the use of the current as an agent for executing criminals has furnished excellent opportunities for the systematic study of the subject.

Pathology.—Some of the lesions are purely surgical, such as the burns which are caused by immediate contact with a "live" wire. It is impossible to state in scientific terms what is the exact pathology of electric shock, especially in cases of sudden death. The results of examinations are often negative. Van Gieson, in autopsies on the bodies of criminals, found fluid blood, but no recognizable changes in the tissues or organs.

Symptoms.—Macdonald and Ward have recorded the effects as noted in the execution of four criminals at Sing Sing, N. Y.[1] With a current of 1785 volts, passed through wet sponge electrodes from the forehead to the calf of one leg, the heart continued to beat after the first contact of 27 seconds, and a noisy respiration was re-established after an interval of more than one minute. After a second contact of 26¼ seconds respiration and the heart action had ceased permanently. It seems that the action of the heart is not permanently arrested as quickly as is respiration. With a stronger voltage, as in a lightning stroke, it is possible that the respiration and the heart are arrested instantaneously.

E. A. Spitzka, whose observations are based upon thirty-one electrocutions, finds that "the death is undoubtedly painless and instantaneous. The vital mechanisms of life, *circulation* and *respiration*, cease with the first contact. Consciousness is blotted out instantly and the prolonged application of the current as it is usually practised by Mr. E. F. Davis,

[1] Medico-Legal Journal, vol. ix. Also XX. Cent. Pract., vol. iii. pp. 403–411.

the State electrician of New York, ensures the permanent derangement of the vital functions so that there could be no recovery of these. Occasionally, the drying of the sponges through undue generation of heat causes desquamation or superficial blistering of the skin at the site of the electrodes, but not often. Post-mortem discoloration, or lividity, often appears during the first contact. The pupils of the eyes dilate instantly and remain dilated in death."

Diagnosis.—This must depend largely, if not entirely, upon the history. Burns on the surface of the body, as already said, are common from contact with a "live" wire, but in the case of lightning stroke, while not unseen, they do not appear to be so extensive or so common.

The after-effects in non-fatal cases usually consist in states of neurasthenia and traumatic hysteria. Organic palsies, or permanent lesions of any kind, seem to be rare sequels.

VII.

THE DIAGNOSIS OF PREGNANCY.[1]

Under ordinary circumstances the question as to the existence of pregnancy, as it presents itself to the general practitioner or to the specialist in midwifery or in gynæcology, presents no difficulty. As a rule, the patient has already made the diagnosis for herself. As it occurs, however, to the medical diagnostician, it frequently assumes a high degree of importance and involves responsibility of the gravest kind. This is especially the case in illegitimacy in young girls, who very often stoutly deny exposure to the possibility of such a condition until the approach of actual labor. On the other hand, married women long childless sometimes positively assert that their hopes are about to be realized, and enumerate in detail and with precision the signs of the condition even to the motion of the child, under circumstances in which the occurrence of such an event is impossible—*pseudocyesis*. There are obvious reasons why the opinion of the medical man is often first sought.

Early Gestation.—**Symptoms.**—Amenorrhœa in a healthy woman previously regular is a symptom of primary importance, and usually the first to arouse a suspicion on the part of the woman as to her condition. Cessation of the menses, under these circumstances, is physiological and unattended by the signs of the grave chronic diseases, as nephritis, tuberculosis, and the cachexias, in which it is pathological. Abrupt cessation at a later period of life may also be physiological and mark the occurrence of the grand climacteric. The occasional occurrence of this physiological event at an unusually early period of life is to be borne in mind. On the other hand, the recurrence of a menstrual flow for two or three months, or in extremely rare instances throughout pregnancy, must be considered in a doubtful case. Nausea is next in importance. It begins, as a rule, from two to four weeks after amenorrhœa and may or may not be associated with vomiting. It is troublesome and distressing at the hour of rising,

[1] Contributed by Dr. W. R. Wilson as collaborator.

but the gravida may be annoyed by waves of nausea from time to time during the day. Cravings for unusual articles of food, and hysterical manifestations, may accompany the nausea. Constipation is common. Further subjective manifestations are irritability of the bladder, increased flow of saliva, and sensations of fulness and tingling in the breasts.

Signs.—Certain signs usually show themselves during the first two months, and have a diagnostic value. Those relating to the breasts comprise general enlargement, a nodular fulness in the glandular area, slight prominence of the nipple, pigmentation of the areola, and enlargement of the sebaceous glands surrounding it. Pressure of the contents of the ducts outward toward the nipple reveals the presence of a yellowish watery fluid—*colostrum*. In multigravida a persistent secretion may follow lactation and simulate colostrum. Signs apparent upon examination of the abdomen are pigmentation of the linea alba, flattening of the hypogastrium, and retraction of the umbilicus. In fair women pigmentation may not occur, and in fat women flattening and retraction are not marked.

Pelvic signs are more distinctive. Of these the more important are a violaceous coloring of the cervical portion of the uterus, thinning and compressibility of the junction of the cervix with the body,—*Hegar's sign*,—lateral expansion of the fundus in anteflexion, and pulsation of the uterine arteries. The body of the uterus presents a peculiar softness. The presence in the os of a plug of tenacious mucus is, in connection with the foregoing changes, very suggestive.

Advanced Pregnancy.—A tendency to constipation persists and waves of nausea occur. Striation of the breasts, abdominal walls, and upper parts of the thighs becomes conspicuous. There are elevation of the fundus uteri and protrusion of the navel. Recurrent rhythmical contractions of the uterus after the completion of the third month, unattended with pain—*Braxton Hicks's sign*—are highly suggestive of pregnancy. Finally, the rise and fall of the fœtus in ballottement, the movements of its limbs, and the sounds of its heart are positive and conclusive signs.

Diagnosis.—The direct diagnosis rests upon the presence of the above symptoms and signs and becomes probable in proportion to the number of them in association at the time of the examination. It assumes greater certainty as pregnancy advances, and demonstrable signs, which offer more positive indications, become associated with the symptoms.

Differential Diagnosis.—*The Distinction between Normal Pregnancy and Conditions which Simulate it.*—As to special symptoms, the amenorrhœa of pregnancy may usually be distinguished from pathological amenorrhœa by the anamnesis. The absence of a history of disease, such as malaria, nephritis, an acute or chronic infection, is important. But the fact that women subject to such diseases may become pregnant is not to be overlooked. Amenorrhœa due to local pathological conditions—hæmatometra, pyometra, and hydrometra—is attended by uterine colic, and the enlargement of the organ does not present the softening of the pregnant uterus. On the contrary, it offers a tense, fluctuating tumor. The amenorrhœa of acquired atresia may be readily recognized by the history and local conditions. The amenorrhœa of the menopause is rarely abrupt, mostly partial and progressive, and frequently accompanied by

hysterical manifestations. Accumulations of omental fat or abdominal-wall fat are common. Moreover, the uterus is not enlarged or softened, and the other local signs of pregnancy recognizable upon vaginal examination are wholly absent.

Intra-abdominal tumors arising from the level of the pelvis are to be distinguished from the gravid uterus by their consistency, as in the case of fibromyomata, where the enlargement is tense, nodular, and of slow growth. The impairment of the patient's health, and the attacks of pelveoperitonitis occurring with more or less regularity, together with the metrorrhagia, are conclusively opposed to the diagnosis of pregnancy. Tumors of the adnexa, and pelvic exudates, reveal their character both by the local symptoms and by the situation in the pelvis of the swellings to which they give rise.

Subjective manifestations of pregnancy which simulate functional disturbances arising in disease, such as nausea, and albuminuria, are to be weighed relative to their association with other symptoms and with the signs of pregnancy. The presence or absence of casts, the amount of urine excreted, and the history of the case render it possible to differentiate the albuminuria of pregnancy from that occurring in nephritis.

Abnormal Pregnancy and Simulative Conditions.—Abnormal pregnancy comprises, first, the development of the ovum in abnormal situations; second, pregnancy following the fertilization of the ovum *in loco* but pursuing an abnormal course.

As to the points of difference in the diagnosis of tubal gestation and cornual pregnancy from pelvic tumors simulating either of these conditions, the irregular uterine bleeding, the pain, the collapse occurring with rupture or tubal abortion, and the symptoms of internal bleeding, together with the localization of a mass outside the uterus—all in the presence of symptoms of early pregnancy—are suggestive.

Certain symptoms, which by their occurrence characterize the course of pregnancy as abnormal, may give rise to confusion. The continuance of menstruation, for instance, may obscure the diagnosis. The syncope which occurs in pregnancy may likewise require the observation of associated symptoms to explain its presence. The œdema of pregnancy shows itself in the presence of signs which distinguish it from the œdema of cardiac disease and the anasarca of nephritis; it is usually progressive without symptoms of nephritis, is not accompanied by pallor or waxiness of the skin, and is evidently connected with the pressure of the uterus.

Finally, hydramnios may suggest the presence of a tumor within the abdomen originating from some condition other than pregnancy. Hydramnios, however, may be diagnosticated by the presence of fluctuation, the rapid increase in the size of the tumor, the location of the latter within the abdomen, and the associated symptoms and signs of pregnancy.

Serodiagnosis of Pregnancy—Improved Method of Abderhalden.—This laboratory procedure depends upon the principle that the blood-serum of pregnant individuals contains a specific ferment which digests placental cells.[1]

[1] Consult Müller's "Serodiagnostic Methods," Translated by Ross C. Whitman, p. 68, *et seq.*, J. B. Lippincott Company.

VIII.

THE DIAGNOSIS OF THE RHEUMATOID AFFECTIONS.

I. ARTHRITIS DEFORMANS.

Definition.—A chronic disease of the joints, of undetermined causation, characterized anatomically by lesions of the synovial membrane with hypertrophy of its fringes, atrophic changes in the cartilages and bones, irregular hypertrophy of the bones, and wasting in the periarticular structures; and clinically by characteristic deformities.

Etiology.—Predisposing Influences.—The synonyms *rheumatic gout* and *rheumatoid arthritis* indicate the prevalent belief that arthritis deformans has some relationship to those affections—a belief that finds little support in fact. In less than one-third of the cases the family history shows a tendency to joint disease—gout or rheumatism. Arthritis deformans in successive generations is unusual. Two or more cases have been noted in a family. Children are sometimes affected; young girls frequently; the greater number of cases begin in early adult and middle life. Women are affected more frequently than men. The disease very often first shows itself about the time of the menopause, and is more common among women who suffer from diseases of the reproductive organs. Habitual exposure to cold and damp, hardship and privation, sudden mental shock, and depressing emotions, appear to bear a causal relation to the disease.

Exciting Cause.—There are two theories: first, that the joint affection is secondary to some disease of the nervous system; second, that it is a chronic infection. The latter is now generally accepted. A variety of micro-organisms have been found in the lesions, but none that is uniformly present. The fact that, in a considerable proportion of the cases, there is a history of gonorrhœa, does not, in view of the wide prevalence of that disease, indicate a causal relation on the part of the gonococcus.

Morbid Anatomy.—All the tissues which enter into the structures of the affected joints are involved in the morbid process, but in which of them the process starts cannot be affirmed with certainty. It is probable, however, that the cartilages are first affected. The lesions consist of fibrillation and atrophy of the cartilages; in the bones, eburnation, abrasion, and osteophyte formation; in the synovial membranes, thickening and hypertrophy of the fringes and atrophy of the periarticular tissues. Osteophytes developing at the margins of the bones may interfere with movement. Bony ankylosis is rare in the joints of the extremities, but common in the spine, which sometimes becomes rigid and immovable. A late condition is contracture, with fixation of the joints in flexion. On the other hand, the muscles are atrophied and the ligaments relaxed, so that subluxation is common, especially in the knees and fingers. The hands are greatly deformed and the fingers, under the influence of gravity, show deflection to the ulnar side. Some of the joints are the seat of an

effusion. Neuritis occasionally occurs. The most striking feature of the arthropathy is its symmetry. The changes in the cartilages and bones are well shown in radiographs.

Clinical Varieties.—Heberden's nodes; the progressive polyarticular form; the monarticular form; the vertebral form; and arthritis deformans in children.

(a) Heberden's Nodes.—"Tumors attaining to the size of a pea, which are sometimes developed near the third joints of the fingrs. They have certainly nothing in common with arthritis (gout), since they are met with in many persons to whom that disease is unknown. They remain throughout life, are devoid of all pain, and show no tendency to ulceration. The deformity is more conspicuous than the inconvenience they cause, though the movement of the fingers is somewhat impeded by them." They occur much more commonly in women than in men, and about middle life. The nodules may become tender and red. Tophi do not appear. The larger joints are not involved. They are not influenced by treatment.

(b) The Progressive Polyarticular Form.—The *acute variety* in the initial attack bears a close resemblance to rheumatic fever, for which it is very often mistaken. It occurs frequently in young women among the working classes, and especially in mill girls. It is common after childbearing and during lactation. It begins in some of the cases at the menopause. The joints become swollen, tender, and painful upon movement; there is fever and the patients become anæmic and weak, and rapidly lose flesh. The attack passes over, leaving the affected joints slightly deformed. From time to time similar attacks recur, each leaving, as it subsides, some increase of deformity and further impairment of health, until at length the patient becomes completely broken down and crippled. The small joints of the hands and feet first and chiefly suffer, but the ankles, knees, wrists, elbows, shoulders, and spine frequently become involved. *Chronic Form.*—This variety is most common. Acute attacks may occur. Only one or two joints may be at first affected; usually the hands are first involved, then the knees or feet. Gradually new joints suffer until, in the severe cases, scarcely an articulation escapes. As the disease progresses the joints are symmetrically involved. The earliest symptoms are pain on movement and slight swelling, which may be intra- or periarticular. The pain varies greatly in intensity. There are cases in which a high degree of deformity gradually comes to pass, without pain; others in which there is pain only at the time of outbreaks of the arthritis, or at night; and a few in which pain is intense and persistent.

The deformities are progressive. The joints are enlarged in part by the outgrowth of osteophytes, in part by thickening of the capsular ligaments, and in part by subluxation. While they remain movable, crepitation may be felt. The periarticular tissues, and especially the muscles, undergo atrophy, and at length the function of the joints is wholly lost. Osteophytes, adhesions, and infiltration of the tissues prevent movement, and the joints become fixed, usually in strong flexion, so that in extreme cases the patient lies completely helpless, unable to move any part of the

body except the eyes. Trophic changes, atrophy of the muscles, glossy skin, pigmentation, and onychia occur, and numbness and tingling are common. In many of the severe cases the joints of the hands and wrists suffer to a less extent, and the ability to sew or write is in part retained. A considerable proportion of the cripples caused by this form of arthritis deformans maintain fair general health and a cheerful disposition.

(c) The Monarticular Form.—This form is usually seen in elderly persons. The knee, hip, and shoulder are commonly involved. They very often follow traumatism. In other cases they develop insidiously. Exposure to damp cold appears to exert a predisposing influence, and aged fishermen, oystermen, and hunters often suffer. The joint lesions are the same as in the polyarticular forms; the muscles rapidly waste, adhesions limit the movements of the parts, and motion is extremely painful.

(d) The Vertebral Form.—Two varieties are recognized. In one, the spine alone is involved. The disease begins with obscure meningeal symptoms, with evidences of compression of nerve-roots, pain, anæsthesia, loss of function, and wasting of spinal muscles, atrophy of the disks, and progressive ankylosis of the vertebræ (Von Bechterew). In the second variety, the hips and shoulders are also ankylosed—spondylose rhizomélique—(Strumpell-Marie). Spondylitis deformans is more common in males than in females. It may result from spinal injury. The early manifestations are sometimes confined to the cervical or to the lumbar region. In other cases the entire spine gradually becomes rigid and immobile. There may be marked kyphosis, with a rigid and immobile thorax and with diaphragmatic breathing.

(e) Arthritis Deformans in Children.—The disease occurs in early childhood. Girls are more commonly affected than boys. The disease may present the same features as in adult life. In some cases direct inheritance has been noted. Cold, privation, and unsanitary surroundings are predisposing influences. The onset may be acute, with the symptoms of infection, fever, profuse sweating, enlargement of the spleen and superficial lymph-glands—*Still's disease.* One or two joints may be at first affected, and others later. There is loss of function together with muscular atrophy. The prognosis is more favorable than in adult life.

Diagnosis.—The direct diagnosis depends upon the subacute exacerbations of the joint affection, the fact that after each outbreak the deformity is increased, the remarkable symmetry of the lesions, the persistence of the process in the affected joints, the relaxations of the ligaments, the atrophy of the muscles, and the progressive character of the disease. The initial attack often presents a remarkable resemblance to subacute rheumatic fever.

Differential Diagnosis.—From rheumatic fever the diagnosis may be made by the permanence of the arthritis in particular joints, the persistence of the lesions, the extreme infrequency of endo- or pericarditis, and the incurability of the affection; and from gout by the history, the fact that gout is mostly a disease of men and middle life, and the absence of tophi. The monarticular forms are usually regarded as cases of chronic rheumatism.

Most of the cases of so-called chronic rheumatism are forms of arthritis deformans, especially those in which a single joint is involved. A majority of the cases of so-called muscular rheumatism are purely myalgic. Nevertheless, out of deference to an almost universal custom, chronic rheumatism and muscular rheumatism are permitted to retain their nosological position. It is important to note the entire absence of etiological and clinical relationship to rheumatic fever.

II. CHRONIC RHEUMATISM.

Definition.—A chronic joint affection of elderly persons, characterized anatomically by synovial inflammation, capsular thickening, and wasting of the periarticular tissues and the related muscles, and clinically by stiffness, pain, and impairment of motion.

Etiology.—Predisposing influences are advanced age, occupations which expose the individual to cold and damp—as in the case of washerwomen, ditch-diggers, and fishermen—poverty, and hardship. The exciting cause is sometimes an injury, which may be slight. In many cases the disease develops insidiously.

Morbid Anatomy.—The synovial inflammation is unattended by effusion. There is thickening of the ligaments, especially of the capsular ligament and the sheaths of the tendons. Erosion of the cartilages may be found. There is marked atrophy of the muscles related to the affected joint. Subluxation and other deformities are usually not marked until late in the course of the disease.

Symptoms.—Pain, which is more marked in the morning and when the weather changes; tenderness, which subsides after gentle massage; stiffness, which is usually relieved to some extent by exercise, are the chief symptoms. Swelling is commonly present, but not redness. A single joint is often affected, as a knee, hip, or shoulder; in many cases several joints are involved, more commonly the large than the small joints. Ankylosis and more or less deformity may gradually come to pass. The subjects are often broken down and anæmic. The prognosis as regards cure is not hopeful.

III. MYALGIA.

Muscular Rheumatism.

Definition.—An affection of the voluntary muscles and their fibrous structures, of undetermined pathology, characterized by pain upon movement and pressure. The disease is local and is designated by various names, as lumbago, torticollis, pleurodynia, according to the parts involved.

Etiology.—Predisposing Influences.—The rheumatic and gouty habit of body, laborious occupations, and those involving exposure to cold and damp are important factors. Men suffer more frequently than women. It is an affection of middle and late life.

Exciting Causes.—The attacks follow cold and exposure, especially when heated. A draught of air may bring it on. Overuse of a group of

muscles is a frequent cause. The muscular pains and soreness after a first horseback ride are myalgic.

Whether the pain and tenderness arise from some nutritional disturbance of the muscle substance acting upon the sensory nerves of the muscles, or these symptoms are due to a neuralgia of such nerves, has not been positively settled. Myalgia is usually acute; it may be subacute and is sometimes chronic.

Symptoms.—The disease is local. Constitutional derangements are rare and due to pre-existing or accidental conditions. They consist of loss of appetite, languor, and slight rise of temperature. Pain is the chief symptom. It is rarely constant, but is acute, even agonizing, when the affected muscles are contracted. It is sharp and cramp-like upon movement, but dull and sore, or absent altogether, when the muscles are in repose. Firm pressure causes soreness, as may be seen in lumbago—a sign of diagnostic value.

Fig. 307.—Acute rheumatic torticollis.—Rotch.

According to the seat of the affection the following principal varieties are described: Lumbago.—The erector muscles of the spine and their attachments are affected. There is pain upon rising and turning. The patient can lean over to lace his shoes, but cannot straighten his back without pain. There is marked tenderness upon firm pressure over the affected muscles and their attachments. The attack is of sudden onset and often completely disabling. Torticollis—Wry-neck.—The sternocleidomastoid and adjacent muscles are affected. In some instances the posterior cervical muscles are also involved. This form is very common. Young persons frequently suffer. The head is held rigidly and cannot be rotated from side to side. Pleurodynia.—The intercostal muscles and, in some cases, other chest muscles are painful and tender upon pressure. The left side is more commonly affected than the right. A deep breath, coughing, sneezing, even laughter may cause the patient to cry out with pain. It is to be distinguished from pleurisy by the absence of friction râles and from intercostal neuralgia by the absence of painful points along the course of the nerves, and the fact that in the latter affection the pain is more paroxysmal. The epigastric pain often seen in measles is myalgic. Other forms are cephalodynia, in which the muscles of the scalp are involved; dorsodynia, scapulodynia, and so on.

IX.

THE DIAGNOSIS OF DISEASES DUE TO DISORDERS OF METABOLISM.

I. GOUT.

Podagra.

Definition.—A disease of disordered metabolism, due to the presence in the blood of uric acid in abnormal amounts, and characterized clinically by attacks of acute arthritis involving one or several joints and recurring at irregular intervals, the deposition of sodium biurate in and around the joints, and irregular constitutional symptoms.

Etiology.—It is necessary to consider the gouty constitution and the attack. An individual may present marked evidences of the gouty diathesis and never experience an attack; or there may, fortunately, be long intervals between the attacks, during which the health is excellent. As the disease advances the attacks become more frequent and the general health is progressively impaired.

Predisposing Influences. — *Heredity.* — The gouty constitution is inherited more frequently than it is acquired. Transmission appears to be more common in the male line, but in this connection the different mode of life of the sexes is to be considered. In hereditary cases the attack may occur in childhood. It is not common before thirty, but in a majority of the cases the first attack occurs in early middle life. Alcoholic excesses, and especially the habitual free use of alcoholic beverages—*regular drinking*—is an important causative factor. Malt liquors tend to cause gout more than wines, and these more than distilled spirits. *Excesses in food* are more important still. Eating too much, without active exercise, is a predisposing cause of the highest importance. Gouty persons are often hard workers, with excellent appetites and good gastric digestion. Moreover, rich viands and fine wines are associated in the pleasures of the table. But gout is not restricted to the rich; exhausting toil, poor food, and excesses in beer may cause it—"*poor man's gout.*" *Chronic lead poisoning* plays an important part in the etiology of this disease. *Sedentary Habits.*—Lack of fresh air and sunshine, with prolonged mental effort, predisposes to the attack. Sydenham's "Whenever I return to my studies my gout returns to me" is well known.

Exciting Causes.—When the attack is due, it may be brought on by apparently trifling causes—an unusually hearty meal, a glass of champagne, sudden chilling, worry, or sudden depressing emotion, or mental shock. It may also be precipitated by a slight injury or accident.

Pathology.—The nature of gout, beyond that it is due to faulty metabolism with overproduction and deficient elimination of uric acid, is unknown. The hypotheses regarding the actual part played by uric acid, which is now regarded as one of the purin bodies derived from the nuclein resulting from nuclear disintegration, in causing gout are very numerous. None of them, however, arises to the dignity of a theory.

Symptoms.—Gout may be acute, chronic, and irregular.

1. Acute Gout.—The attack is commonly preceded by prodromes, which consist of fleeting pains in the small joints of the hands or feet, restlessness, irritability, and dyspepsia with acid eructations. In some cases there is an erythematous angina, bronchitis, or asthmatic symptoms. The elimination of uric acid has been found to be diminished before and during the early part of the attack. In many instances there are no premonitory symptoms. The attack begins, as a rule, in the early morning hours. There is agonizing pain in the metatarsophalangeal joint of the great toe, more frequently the right, or the tarsometatarsal joints, especially at the outer border of the foot. There is intra- and periarticular effusion; the skin is hot, red, tense, and glistening. Tenderness is extreme and the patient cannot endure the slightest pressure upon the affected part. There is moderate fever, the temperature rising to 102°–103° F. (39°–39.5° C.) and falling to normal toward the end of the attack by lysis. The intensity of the symptoms somewhat abates in the morning, but the foot remains swollen, red, and painful, and during the night the patient's sufferings are again intensified. Other joints, and particularly the great toe of the opposite foot, may become affected. The nocturnal exacerbations gradually subside and, in the course of a few days or a week, the acute symptoms disappear with itching and desquamation, but the affected joint remains tender and swollen, often requiring the use of a loose shoe and crutches for another week or more. Suppuration in the affected joints does not occur. Transient albuminuria or glycosuria may occur. There is a moderate leucocytosis during the acute symptoms. Physical and mental depression persist for a period, but after a time the patient regains his former health. Recurrences follow at intervals varying from a few months to a year or more. In the later attacks not only the joints of the feet, but also the knees, wrists, and fingers may be involved.

Retrocedent or Suppressed Gout; Visceral Gout.—These terms are used to designate groups of symptoms, usually grave, indicative of disease of internal organs, which sometimes arise coincidently with a sudden subsidence of the local manifestations of the attack. The principal groups are, (a) gastro-intestinal—pain, vomiting, purging, and collapse, so severe in some instances as to prove fatal; (b) cardiac—dyspnœa, precordial pain and distress, arrhythmia, and tachycardia; and (c) delirium, stupor, coma, or apoplectiform attacks. These symptoms are in many of the cases uræmic.

Chronic Gout.—The attacks become more frequent and prolonged, and many joints are affected. In debilitated persons and those suffering from chronic lead poisoning, the attacks of arthritis may not occur—*atypical gout.* Deposits of sodium biurate—*tophi*—occur in the cartilages and ligamentous structures of the joints, which, in the course of time, become enlarged and deformed. The feet are first and most markedly affected, then the hands to a less extent, and in some cases tophaceous deposits form about the knees and elbows or in the line of the tendons. The cartilage of the ear frequently contains tophi which appear as yellowish-white nodules at the edge of the helix. Less frequently similar deposits take place in the cartilages of the nose, eyelids, and larynx. The skin covering

the tophi frequently undergoes slow necrotic change, with ulceration exposing the felted chalk-stones. As the disease advances the patients become sallow and dyspeptic; the signs of arteriosclerosis develop; there are cardiac hypertrophy, increased arterial tension, increased urine with low specific gravity, slight albuminuria which may be intermittent, and hyaline casts. Muscular cramps, especially affecting the calves of the legs and starting in the predormitium, are common. Attacks of arthritis, implicating one or several joints, characterized by pain, redness, and swelling, occur with or without fever. Croupous pneumonia and apoplexy are common terminal events, but death often results from uræmia or from an acute inflammation of one of the great serous sacs.

Irregular Gout.—In addition to the attacks of arthritis and the chalk-stones, there are diverse morbid conditions to which gouty subjects and members of gouty families are alike especially liable, and which are commonly regarded as irregular manifestations of gout. Among the more important of these are cutaneous eruptions, and especially eczema; periodical gastro-intestinal catarrh—so-called bilious attacks; pulmonary affections, especially bronchitis and emphysema; cardiovascular lesions, myocardial degenerations, arteriosclerosis, and aneurism and nervous affections, among which migraine, headache, sciatica, and other neuralgias are the more common. Burning sensations and itching of the feet at night are very common and annoying symptoms. Among affections of the eye, scleritis, iritis, cataract, glaucoma, and hemorrhagic retinitis have been attributed to gout. It is the custom to regard gout as the cause of the recession of the gums so common in advancing life.

The urinary conditions are more especially gouty because they are based upon anatomical lesions of the kidneys. Chronic interstitial nephritis, without characteristic changes, is not uncommon in gouty subjects. More common are conditions believed to be distinctive, namely, a deposit of urates in the intertubular tissue, mostly in the papillæ; less frequently the deposits occupy both the tissue and the tubules, and there are also minute foci of necrosis in the cortex and medulla, in which are deposited crystals of sodium urate. The clinical manifestations of gouty deposits are the same as those of this form of chronic nephritis occurring in non-gouty persons: increased output of urine, albuminuria (usually slight), low specific gravity, hyaline casts, increased arterial tension, accentuated aortic second sound, and uræmic symptoms. Prior to the development of renal changes the urine is often very acid and high-colored and may, upon standing, deposit crystals of uric acid. In chronic gout the uric acid is diminished, as a rule, with occasional excess. Intermittent glycosuria, traces of albumin, and tube casts may occur. Oxaluria is common. Renal calculi are not infrequent. The association of vesical calculi and gout is often observed. I have several times seen purulent urethritis follow an attack of gout.

Diagnosis.—The direct diagnosis of acute gout is usually a simple matter. Recurrent attacks of arthritis, beginning in or limited to the great toe, with a hereditary history of gout and a personal history of overindulgence in food and drink, are of positive diagnostic value. When other joints are implicated and there is fever, and a satisfactory family

and personal history cannot be obtained, there may be a question as to the differential diagnosis between acute gout and rheumatic fever. The following facts are in favor of the diagnosis of gout: the occupation and habits of the patient; the involvement of a limited number of larger joints; the persistence of the arthritis in the affected joint in contrast to the migratory character of rheumatic arthritis; the appearance of the inflamed joints, which are tense, deeply red or violaceous, and shiny; the suddenness of onset; and the condition of the urine, which shows a low uric acid output in the beginning of the attack, with marked increase toward its close. The examination should be made under a purin-free diet.

The Diagnosis of Chronic Gout. — The history, the presence of tophi, and the deformities are characteristic. A tophus in the neighborhood of a joint is easily recognized. Tophi upon the ears appear earlier and are positively diagnostic. Other things at the ear margin may be mistaken for them: the helical apex, called from the English sculptor and poet Woolner's tip; small sebaceous tumors; and fibroid nodules. The last are very rare. In the felted material from an open tophus the needle-shaped crystals of sodium biurate are characteristic. Garrod's uric acid thread test may be tried. In a watch glass, 15 c.c. of blood-serum, which may be obtained by blistering, are treated with 0.25 of acetic acid. A fine thread immersed in it may show in a few hours crystals of uric acid. The result is often negative in cases in which there is no question as to the clinical diagnosis. An excess of uric acid in the circulating blood occurs also in leukæmia and chlorosis.

The differential diagnosis between chronic gout and arthritis deformans demands some words of consideration. This necessity arises more from the misleading influence of the term "rheumatic gout" than from any real resemblance between the diseases. In the rare cases in which the deformities of chronic gout arise insidiously, in the absence of acute attacks, the following points are important: Arthritis deformans is common in women and among the poor and poorly nourished; spontaneous pain in the affected joints is less common and less urgent; the deformities, especially those of the hands, are more uniform and symmetrical; and tophaceous deposits form no part of the pathology of the disease.

The diagnosis of irregular gout is based upon the family and personal history of the patient, and the general experience of clinicians that gouty individuals more frequently manifest these particular derangements of health than others. The diagnosis of visceral gout depends, likewise, upon the anamnesis and the fact that the onset of the symptoms, indicating disease of a particular organ, corresponds in time to the subsidence or disappearance of the familiar symptoms of the acute attack of gout. The diagnosis of irregular, retrocedent, and visceral gout should be made with some reserve and only in the case of a distinct hereditary predisposition, or of an individual who has had acute attacks, or who shows tophi or characteristic deformities, and in whom, in the absence of such signs, other etiological factors can be excluded.

II. DIABETES MELLITUS.

Definition.—A chronic disorder of metabolism due to diminished capacity for the combustion of carbohydrates, and characterized by the persistent excretion of glucose in the urine when moderate amounts of carbohydrates are ingested, or even none at all in certain cases. Polyuria, polydipsia, polyphagia, and emaciation are prominent but not constant symptoms.

Diabetes mellitus and diabetes insipidus have little in common except a persistent increase in the amount of urine secreted. Glycosuria, a symptomatic condition characterized by a transient presence of sugar in the urine, corresponds to polyuria, a symptomatic condition in which the urinary output is greatly increased for a short time. These conditions are to be distinguished from diabetes mellitus and diabetes insipidus, which are substantive diseases.

Etiology.—Predisposing Influences.—Diabetes mellitus prevails in every part of the world, but more extensively in some countries than in others. Southern Italy and India suffer to an especial degree. The disease is about as prevalent in the United States as in Europe and appears to be increasing upon both sides of the Atlantic. In all countries it is more common among those living in affluence than among the poor. The Semitic race manifests an especial predisposition to the disease. This racial peculiarity has been noted by competent observers in various countries. In the United States the negroes suffer to a less extent than the whites.

The predisposition is very commonly inherited. The remarkable prevalence of diabetes among the Hebrews is in many of the cases due to this tendency. Not only is the disease observed in successive generations in the direct, but also in collateral, lines, and it occasionally occurs at an early age in two or more children of the same family. The descendants of gouty or obese persons show an especial liability to diabetes. R. Schmitz first directed attention to the possibility of the transmission of the disease from one person to another. A long and intimate association, as in the case of a wife taking care of a husband suffering from the disease, has, in rare instances, been followed by the development of the same symptoms. Previous good health on the part of the second individual, with absence of hereditary predisposition, has been established in those cases. They are extremely rare and the transmissibility of the disease appears highly improbable.

Diabetes mellitus is more common in men than in women, the ratio, according to available statistics, being about three to two. The disease may occur at any period of life. Infants at the breast are sometimes affected, but such cases are extremely rare; they also run a rapid and fatal course. Hereditary influences are usually in evidence, and several of the children in one family may be affected. But diabetes mellitus is essentially an affection of adult life. A majority of the cases come under observation between the third and sixth decades. The disease is often discovered upon routine medical examination for life insurance or other purposes, and has already existed in many cases for a considerable time. Those who live luxurious, aimless, and idle lives are peculiarly liable to

the disease. The wear and tear of a strenuous intellectual life, especially when coupled with great anxiety and mental excitement, contribute a predisposing influence of great importance. Absorbing application to business, excesses at table, and a sedentary life are important factors in producing the disease. Those who dwell in cities suffer in greater proportion than countrymen. It is, however, to be noted in this connection that the disease is less apt to be recognized in the latter class. Neurotic persons are more commonly affected than those of a phlegmatic temperament, a fact to be considered in the matter of the great relative frequency of the disease among the Jews. Gout, syphilis, and malaria have been regarded as predisposing influences. It was at one time thought that the children of phthisical parents were especially liable to diabetes. The disease frequently develops during the course of chronic nervous affections; still more frequently forms of nervous disease, particularly neuralgia, neuritis, and neurasthenia, are dependent upon the diabetes, and arise as intercurrent affections during its course. Diabetes occurs among the insane, but not, according to the statistics of large institutions, in greater proportion than in general hospitals. Obesity is frequently associated with diabetes. In a majority of such cases the obesity precedes the diabetes often by a period of years. Under these circumstances the disease commonly runs a favorable course, the glycosuria diminishing, even disappearing under a moderately strict regimen, and reappearing when the rules are neglected. Much less favorable are the cases in which obesity and diabetes are simultaneously developed in early life. The form that develops consecutively to obesity has been designated "lipogenous diabetes." Von Noorden, who believes that in the obese cases the burning up of sugar is interfered with, and not its conversion into fat, proposes for this form the term "diabetogenous obesity."

Exciting Causes.—*Psychical.*—Mental shock, intense nervous strain, worry, and violent depressing emotions are frequently followed by diabetes. *Physical.*—Disease or injury of the brain or spinal cord, an irritative lesion of the diabetic centre, and epilepsy may also give rise to the disease. The infectious febrile diseases, especially enteric fever, influenza, diphtheria, rheumatic fever, and syphilis, appear in some instances to have been the starting point of diabetes, the symptoms of which have shown themselves either during or directly after the attack. Focal infection particularly in the teeth, gums and tonsils is present in a very large proportion of diabetics. Under all these circumstances the causal importance of the particular event or condition depends upon the known absence of glycosuria prior to its occurrence. The essential relationship between acute and chronic infections has not yet been demonstrated. In the vast majority of cases diabetes mellitus develops insidiously, without discoverable cause.

Pancreatic Diabetes.—It has long been known that diabetes and disease of the pancreas are occasionally associated, and Lancereaux described, in 1877, a special form of diabetes under the name *diabète pancreatique.* The discoveries of Minkowski and von Mering, in 1899, aroused intense interest in this subject. The facts are, first, that experimental extirpation of the pancreas is followed by glycosuria; second, that if a portion of the gland is allowed to remain, glycosuria does not occur; third, that in a considerable proportion of the cases of diabetes, lesions of the pancreas

have been found—sclerosis, chronic interstitial inflammation, hyaline degeneration of the islands of Langerhans; fourth, that the glycosuria is secondary to the lesions of the pancreas. The theory of an internal secretion containing a glycolytic body necessary to the proper combustion of glucose in the muscles supplies the key to the above facts. Pancreatic disease causes diabetes by arresting the formation of the internal secretion of the organ. While by far the greater number of cases are due to disease of the pancreas, there are many instances in which the metabolic fault appears to have its origin in lesions of the central nervous system, the liver, the kidneys, the chromaffin tissue system, the thyroid and the hypophysis cerebri. These structures are sometimes spoken of in this connection as "*diabetogenous organs.*"

Carbohydrate Metabolism.—In health the carbohydrates of the food are stored in the liver and muscles in the form of glycogen. This substance is also formed from the proteids of the food, and under certain conditions glucose is formed from the proteids of the tissues of the body. The glycogen is again converted into glucose and given up gradually to the blood, in which it circulates in a 0.1 to 0.2 per cent. solution, to be distributed to the muscles, where it undergoes combustion, with the production of heat and energy. According to the investigations of the younger Cohnheim this is brought about by the action of the glycolytic bodies, one derived from the muscles, the other from the pancreas.

Whenever the glucose in the blood is in excess of 0.2 per cent. glycosuria results. This may occur in the absence or in the excess of the glycolytic body: by the sudden ingestion of an excess of carbohydrates—more than 180 to 250 grammes, fasting—*alimentary glycosuria:* and by derangements of circulatory disturbances or instability of the glycogen-storing mechanism. Alimentary glycosuria not only occurs in consequence of the sudden ingestion of large quantities of sugar, but it also appears under certain circumstances after the taking of excessive amounts of starchy food.

Symptoms.—In a large proportion of the cases the onset is insidious and not attended by symptoms which attract the attention of the patient. In some instances, in physicians and others who have examined their urine at intervals, the disease has been preceded by an intermittent glycosuria, which, after a period of months or years, has become persistent. There are other cases in which, under treatment, glycosuria has disappeared, to recur when the strict regimen has been relaxed. The existence of the disease is usually recognized by the occurrence of conspicuous symptoms, as polyuria, polydipsia, polyphagia, emaciation, or pudendal pruritus, or by the discovery of sugar in the urine upon examination as a matter of routine or for life insurance.

In rare instances it follows an injury, profound depressing emotion, or a chill. There are cases in which thirst is not inordinate and the amount of urine not excessive. The tongue in established cases is usually red, dry, and denuded of epithelium: the saliva scanty: and the gums swollen and spongy. There are constipation and lumbar pain. The skin is dry and harsh and perspiration is scanty or absent altogether. In women pruritus vulvæ is a common and distressing symptom, and general pruritus is of

frequent occurrence in both sexes. The pulse-frequency is high and there is increased arterial tension. The temperature is commonly slightly subnormal. Emaciation is common and rapid in young subjects, but older persons may preserve their weight for long periods.

The Urine.—Under a rigorous regimen the quantity may not be greatly increased, and it may be reduced to normal during an intercurrent febrile disease. It is, however, commonly increased to three or four litres in cases of moderate severity, and may reach as much as twenty litres in twenty-four hours in grave cases. The specific gravity ranges from 1.030 to 1.045, but in exceptional cases may be low, 1.015 to 1.020, a fact to be borne in mind in the diagnosis. It is pale in color, with a faint greenish tinge, and has a mawkish, sweetish odor, and is said to have a sweetish taste. Sugar is present in amounts varying from 1.5 to 5 or even 10 per cent. The total quantity excreted in twenty-four hours ranges from 300 to 750 grammes or more.

The studies of Allen (1915) in regard to the management of diabetes do not fall directly within the scope of this volume. They, however, not only revolutionized the treatment of the condition but they also aroused a widespread interest in its natural history and greatly stimulated the scientific study of its phenomena. He holds that diabetes should be regarded as a weakness of a bodily function rather than as a progressive, fatal disease, and that the treatment consists in preventing the patients from overtaxing the weakened function. The plan, which has been called the starvation method, consists in the sudden and complete withdrawal of all food until the sugar disappears from the urine and for 24 to 48 hours longer. During this period alcohol and sodium bicarbonate are given. Food is then administered little by little, the tolerance for carbohydrates being cautiously tested, then that for proteins, then that for fats. Meanwhile the bulk of the food, so necessary to relieve the patients' cravings and prevent constipation is carefully regulated. Under this plan the glycosuria and ketonuria disappear. Should traces of either appear a fast day becomes necessary, after which the tolerance testing is resumed. The patients lose weight, but they feel better and there is a general improvement except in the very severe cases. From time to time single fast days are recommended and a fast day is imperative upon the reappearance of glycosuria or acidosis. Quantitative tests for glucose are unnecessary and the patient himself can readily detect traces of sugar by Benedict's test. Allen believes that under this management early instituted and rigorously carried out severe diabetes mellitus would rarely develop and the type would in most instances continue to be mild.

Tests for Glucose.—The most satisfactory tests for clinical purposes are Benedict's, Fehling's, Trommer's, the bismuth test, the fermentation test, and polariscopy. If close results are desired the chemical tests may be controlled by fermentation or the polariscope. The urea and calcium salts are increased, the uric acid does not show important changes, and the phosphates may be much increased.

Phosphatic Diabetes.—This term has been applied to cases in which there is an excessive excretion of phosphates, with symptoms similar to those of diabetes mellitus but with inconstant glycosuria.

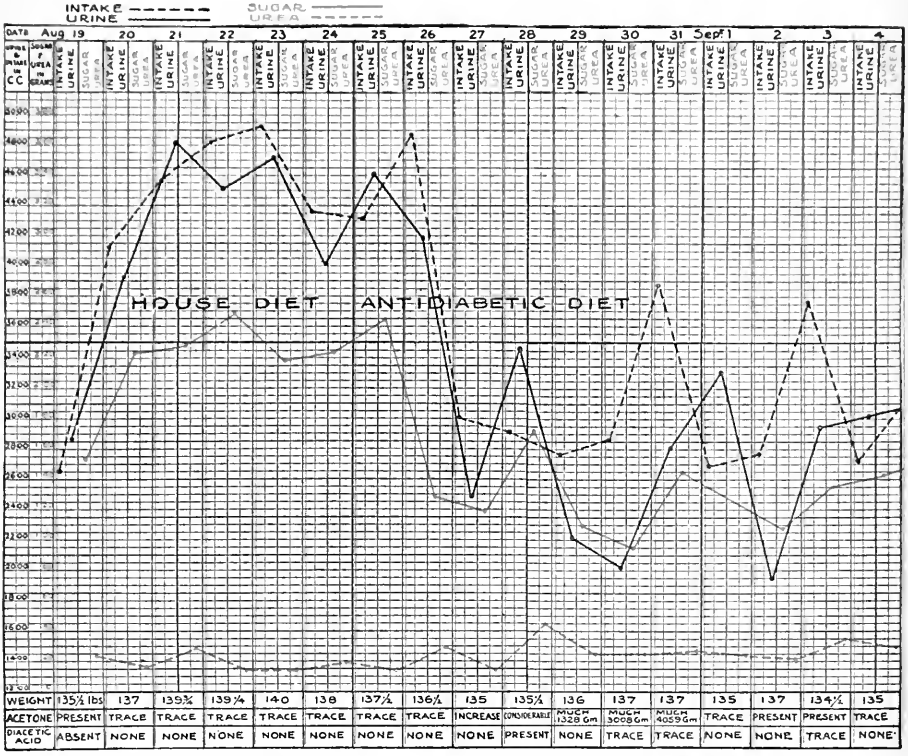


FIG. 308.—Chronic diabetes mellitus; male, age 44. Chart showing daily variations in the intake of fluid, the amount of urine, the quantity of sugar and urea, the specific gravity, and body weight.—Jefferson Hospital.

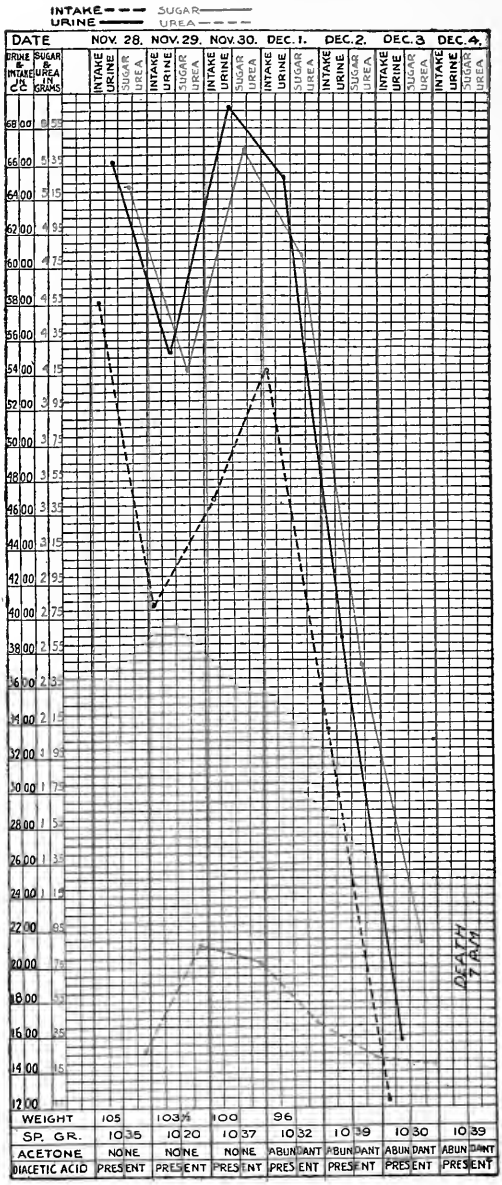


FIG. 309.—Acute diabetes mellitus; male, age 27. Chart showing daily variations in the intake of fluid, the amount of urine, the quantity of sugar and urea, the specific gravity, and the body weight. —Jefferson Hospital.

Acetone and diacetic acid are often present. The presence of these substances in the urine is conclusive evidence that β-oxybutyric acid, of which they are derivatives, is being produced within the organism. There are three stages in the excretion of the acetone bodies: (a) acetone alone in the urine—this substance is exhaled with the expired air; (b) diacetic acid is also present in the urine; and (c) β-oxybutyric acid is present in the urine in addition to acetone and diacetic acid. Much of the acetone fails of excretion by the urine, since, being highly volatile, it passes off by the respiratory surfaces and lends its characteristic odor to the atmosphere about the patient. The presence in the urine of these bodies is of the highest clinical importance, since β-oxybutyric acid is the cause of diabetic coma.

The accumulation of these organic acids in the body constitutes one of the forms of acidosis; their presence in the urine is known as ketonuria.

Glycogen has been found in the urine. Albumin is common, especially in the advanced stages of the disease. *Pneumaturia.*—Gas in rare instances of diabetes passes from the urethra with a bubbling sound. Its presence is the result of fermentative processes in the urine within the bladder. An associated cystitis is common.

The Blood.—Hyperglycæmia is constant. An increase in the cellular elements—polycythæmia—may occur, the erythrocytes reaching 6,000,000 or 7,000,000 per cubic millimetre. In coma there is leucocytosis, and β-oxybutyric acid is present. *Lipæmia.*—Large quantities of fat are often present in the blood. The plasma presents a milky appearance, and if the blood is allowed to stand in a glass a thick creamy layer forms at the top, the nature of which is apparent upon testing with ether, osmic acid, and other substances which possess characteristic reactions with fats. A similar fatty layer is obtained by centrifugalization. The fat particles may be readily seen upon microscopical examination of the fresh blood. The fat is identical with that of the chyle and is therefore probably derived directly from the food. The methylene blue reaction—Williamson, Bremer—is of diagnostic value.

The Course of Diabetes.—The onset is usually insidious and the course chronic. There are cases that begin suddenly with severe symptoms and terminate fatally in a few weeks. Very rarely the acute cases run a favorable course to recovery or assume the chronic type.

The cases may be grouped as mild, well-developed, and severe.

The mild cases are characterized by the rapid disappearance of glycosuria upon the withdrawal of carbohydrates from the diet and an increase of tolerance under careful management. This form is common in persons who become diabetic late in life. The general health is not greatly impaired and death takes place much more commonly from arteriosclerosis, chronic nephritis or phthisis than from coma.

The Well-developed Cases.—The glycosuria is more persistent and tolerance more difficult to establish. The health is manifestly impaired and complications occur. There is a tendency to acidosis.

The Severe Cases.—Glycosuria is uncontrollable by the most rigid restrictions in diet that can be endured without grave loss of weight and strength. Acidosis is severe. The health is seriously impaired. The patient is in imminent danger of coma, which is the cause of death in more than

60 per cent. of the cases. Gangrene is of common occurrence. Death from *phthisis florida* frequently occurs. This form is encountered mostly in children and adolescents, but is not uncommon after the meridian of life is passed.

Complications.—The diabetic has less power of resistance against pathogenic influences than others, and suffers in a remarkable manner from complications. Among these the following are especially important: Skin.—Local inflammations, and in particular boils and carbuncles, are common. These lesions have their origin in cracks or fissures of the skin by way of which infection occurs. Wounds and injuries heal slowly and granulation tissue has an especial tendency to slough. Gangrene is common and is due to arteriosclerosis. In rare instances perforating ulcer of the foot has been observed. I have recently seen symmetrical perforating ulcer of the ball of the foot in a woman aged 52. Eczema may occur. Pruritus is common, and pruritus vulvæ is an early and harassing complication. A balanitis may occur in men. Bronzing of the skin—*diabète bronzé*—is a rare cutaneous manifestation associated with hæmochromatosis. The skin is usually dry and harsh, but profuse sweating may occur in terminal conditions. Xanthoma and purpura occur as in other chronic affections, but have no direct etiological relation with diabetes. The Lungs.—Pulmonary affections are common terminal events. Croupous pneumonia and bronchopneumonia occur as acute complications. Gangrene may supervene and pulmonary abscess has been observed. Pulmonary tuberculosis of the bronchopneumonic type is common. It runs, as a rule, a rapid course. The Kidneys.—The signs of nephritis, albuminuria, and casts very often occur in the later stages of diabetes mellitus. Œdema of the feet and legs may occur, but the polyuria prevents general anasarca. Dropsy may precede coma. Cystitis may occur. The Sexual Organs.—Amenorrhœa may come on early, or the menstrual function may continue more or less regularly to an advanced stage of the disease. Impotence is common and often an early symptom. Conception is not unusual, but is often followed by abortion. Pregnancy is followed by an aggravation of the diabetic symptoms. The Nervous System.—Diabetic coma is the most important of the complications on the part of the nervous system. This affection "represents the culmination of the specific diabetic intoxication" (Von Noorden). It is often preceded by gastric symptoms, as loss of appetite, nausea, vomiting, epigastric tenderness, and constipation, which extend over days or weeks and are followed by mental dulness, drowsiness, and fatigue symptoms. The dulness increases and the patient, though capable of being roused, quickly falls back into stupor, like a drunken man. The respiration becomes full and deep, without being much increased in frequency. The pulse is quickened, but remains strong and full. Cyanosis is not constant. Finally drowsiness gives way to coma, and in the course of twenty to thirty-six hours death terminates the scene. In another group of cases coma suddenly supervenes in persons who, if not well and

Fig. 310.—Diabetic gangrene.

strong, are in their usual health. This fatal complication often follows some unusual bodily or mental effort. Again, coma diabeticum may follow an acute infectious or septic process, or alcoholic intoxication, or ether or chloroform narcosis induced for surgical purposes. The diagnosis may be obscure when the diabetic becomes comatose in consequence of apoplexy or uræmia.

Minor forms of diabetic intoxication may explain the numbness and tingling, which are common symptoms, and the neuralgias, which sometimes affect the upper or lower extremities, sometimes a single nerve-trunk, as the third or the sciatic. Herpes zoster is encountered in diabetes, but not more frequently than in the non-diabetic.

Tabetic symptoms, lightning pains, loss of knee-jerks, and extensor palsies, manifested by a steppage gait, are mostly the manifestations of a peripheral neuritis, though changes in the posterior columns have been described. Paraplegic symptoms are due to neuritis.

The Organs of Special Sense.—Cataract, almost always double, is a late development and occurs in cases in which the glycosuria is of high grade. It develops with great rapidity in young diabetics, but more slowly in the elderly, in which it cannot be differentiated from the senile form. Retinitis, due to associated renal disease, or the ordinary hemorrhagic form, may occur, and optic neuritis followed by atrophy. Sudden amaurosis without ophthalmoscopic changes may come on in the early stages of diabetes. Changes in the organs of hearing, smell, and taste are not common. Otitis media may occur and be followed by mastoid disease.

Diagnosis.—Direct Diagnosis.—The presence of grape-sugar in the urine, continuing for weeks, months, or years, even upon a diet containing carbohydrates in moderate amounts, is the fundamental diagnostic criterion. Of secondary importance in the diagnosis because they are not constant are inordinate thirst, excessive appetite, the exeretion of abnormally large quantities of urine, and loss of weight. Very often all these symptoms are present and give rise to a characteristic clinical picture. The occasional temporary absence of sugar under a diet which does not contain carbohydrates, or during an acute illness, does not constitute an objection to this diagnosis, since such a diet cannot be indefinitely continued, and sugar constantly tends to reappear in the urine when carbohydrates are added, and also reappears upon convalescence from an intercurrent febrile disease.

In the examination of the urine for sugar the total quantity for twenty-four hours is collected, that of the day and of the night separately. Both the percentage and the total quantity of sugar are ascertained. The presence or absence of acetone and oxybutyric acid is also noted and tests for albumin are made. The specific gravity and reaction are recorded.

The quantitative estimation of the ammonia is of great importance, since it permits conclusions in regard to the approximate amount of oxybutyric acid which is excreted at the same time. When more than 2 grammes of ammonia are excreted in twenty-four hours, there is danger of coma. Practically under Allen's method of management the quantitative examination for glucose at stated intervals, which the patient can do for himself, is essential. It becomes important also at times to know the

blood sugar and to determine the degree of acidosis. (See Vol. I, Functional Tests.)

It is to be remembered that the glycuronates, which sometimes precede the excretion of sugar and are frequently associated with sugar in the urine, reduce alkaline copper solutions but do not ferment, and that the homogentisic acid of alkaptonuria reacts to the copper tests but not to Nylander's bismuth test, nor to fermentation. The fermentation test is the most reliable single test, but must, in doubtful cases, be controlled by other tests, since the yeast sometimes contains sugar.

Differential Diagnosis.—*Non-diabetic Glycosuria.* (a) *Transient Glycosuria.*—In rare instances this condition follows concussion of the brain, cerebral apoplexy, severe neuralgias, and profound depressing emotion. The sugar is present usually for a few hours, in some instances for a few days, and shows no tendency to recur.

Under this heading must also be grouped the acute forms of glycosuria, lasting but a few hours, which accompany poisoning by morphine, amyl nitrite, carbonic oxide, chloralamide, and nitrobenzole; the rare glycosuria noted in biliary colic, and that observed in hydrocyanic poisoning. *Phloridzin Poisoning; Renal Glycosuria.*—The sugar is present so long as the administration of phloridzin is continued, alike when carbohydrates have been ingested, or the animal is fed upon a proteid diet or is fasting. This form of glycosuria is not associated with hyperglycæmia.

(b) *Intermittent Glycosuria.*—This condition occasionally occurs in gouty persons and in the non-gouty as the precursor of diabetes. The assimilation limit for carbohydrates may be determined by the administration of 100 grammes of glucose in solution, two hours after a breakfast of a roll and butter with coffee, during a period in which glycosuria is absent. This amount of sugar should not, in a healthy person, cause glycosuria. The excretion of sugar indicates a fault in the storage or metabolism of the carbohydrates. Many cases of diabetes mellitus begin as intermittent glycosuria.

(c) *Alimentary Glycosuria.*—The limit of assimilation for glucose in the healthy subject varies from 120 to 200 grammes in a single dose. If this be exceeded glycosuria occurs, but only a portion of the quantity ingested appears in the urine, the remainder being stored as glycogen in the liver and muscles for future use. The limit for cane sugar is about the same, that for milk sugar much lower, and for maltose in many individuals very low. It is stated by Von Noorden that in some persons half a litre of beer is sufficient to give rise to sugar in the urine and that this fact should be known to avoid the danger of mistaking a harmless symptom for a serious disease. In rare instances glycosuria follows excesses in alcohol.

(d) *Malingering.*—Persons have been known to feign diabetes by dissolving sugar in the urine. The specific gravity is high and the reactions those of cane sugar; but very well-informed patients may use glucose for this purpose. The fraud is, however, easy of detection.

(e) *Glycosuria in Pregnancy and the Lying-in State.*—Sugar occurs under two circumstances: First, the pregnant woman may have been diabetic before conception or may have become so during pregnancy. The

reactions are those of grape-sugar and the significance is unfavorable; secondly, milk sugar may be resorbed from the breast and excreted by the kidneys. This occurs when there is a hypersecretion of milk or, for some reason, such as fissure of the nipple or the removal of the child, there is an interruption of its withdrawal. The reactions are those of lactose and the prognosis is favorable, the sugar disappearing as the secretion of milk is arrested. The condition is not glycosuria but puerperal lactosuria.

Prognosis.—The dangers of diabetes consist in lowered nutrition, diminished powers of resistance to intercurrent diseases, inflammatory and chronic degenerative processes, and, in the severe cases, an abnormal production of acids.

Favorable prognostic indications are onset or recognition of the disease at an advanced period of life, absence of emaciation, the gouty habit of body, the occurrence in the patient's family of other cases running a mild course, slight glycosuria, and tolerance for moderate amounts of carbohydrates. The prognosis is unfavorable when the disease begins at an early age and when other cases of severe type have occurred in the patient's family. Rapid emaciation, grave intercurrent affections or complications, intense glycosuria, and intolerance for carbohydrates are of ominous prognosis. The patient's circumstances and ability to avail himself of favorable personal influences, such as the avoidance of overwork and worry, are very important. Finally, the excretion of ammonia in large amounts and the presence of the acetone bodies in the urine are of immediate gravity, because they are the common heralds of coma.

III. DIABETES INSIPIDUS.

Definition.—A chronic disease characterized by great thirst and the habitual discharge of excessive quantities of urine of low specific gravity.

This affection is a primary or idiopathic disease and is to be differentiated from the transient or persistent hypersecretion of urine, which is symptomatic of certain forms of chronic Bright's disease and some affections of the nervous system.

Etiology.—Predisposing Influences.—Heredity exerts an important influence. Cases have been known to occur in four generations. The disease is sometimes congenital. There is very often a history of chronic disease on the part of the parents of the patient. Diabetes, renal affections, pulmonary phthisis, gout, and rheumatism have been noted. Diabetes insipidus most commonly develops in early life. It is relatively frequent in young children and rarely begins after thirty. It is more common in males than in females.

Exciting Cause.—The actual cause is unknown. The disease usually develops insidiously without assignable cause. It has in some instances been attributed to excessive quantities of water or beer, and has followed an acute infectious disease, especially influenza. In young children malnutrition arising from neglect, insufficient food, and constitutional taint have been assigned as a cause: in older persons acquired syphilis, alcoholism, worry, anxiety, and prolonged exposure to cold.

The frequency of symptoms referable to the hypophysis cerebri in diabetes insipidus has aroused attention. Bitemporal hemianopsis has been observed. The persistent polyuria has been noted in acromegaly and in adiposogenital dystrophy. It has also followed basilar meningitis of syphilitic origin. These cases form a separate group, cerebral diabetes insipidus, and from this point of view may be regarded as symptomatic of derangement of the function of an endocrine gland.

Symptoms.—The two symptoms which are characteristic are an excessive quantity of urine without sugar, and intense thirst.

The Urine.—The quantity voided is enormous. It often reaches twenty or thirty pints in twenty-four hours. Fifty-six pints have been noted. It may even at times exceed the fluid ingested, the difference being made up of fluid withdrawn from the tissues of the body and the food. It is pale in color and limpid, the specific gravity varying between 1.001 and 1.007. The reaction is faintly acid or neutral. The total urinary solids are increased by one-fourth or one-third. *Inosite—muscle sugar*—is occasionally present in small amounts. Albumin is usually absent until late and grape-sugar is occasionally present toward the close, when the symptoms of diabetes mellitus sometimes occur. Thirst.—This symptom is proportionate to the quantity of urine. As much as fifty pints of fluid have been consumed by a patient in the course of a day. There is usually a remarkable thirst for alcoholic beverages. Bulimia.—The appetite is usually enormous and the digestion well performed. These conditions fail, however, in the terminal dyscrasia, when there may be complete anorexia, flatulence, and unmanageable diarrhœa. The patients are often well nourished and healthy looking for years, the main troubles being unquenchable thirst and frequent micturition. Emaciation is an early symptom and becomes, toward the last, extreme. The saliva is scanty, the mouth dry, perspiration slight, and the skin dry and harsh.

Diagnosis.—Direct.—Extreme thirst, excessive habitual secretion of non-saccharine urine of low specific gravity, and emaciation justify the diagnosis of diabetes insipidus.

Differential.—The distinction between the idiopathic disease and symptomatic polyuria is extremely important. The following forms of the latter are to be considered: *Diabetes Mellitus.*—Persistent glycosuria and high specific gravity are important. If sugar appears in diabetes insipidus it is usually in faint traces and transitory. To this statement an exception must be made in regard to the cases in which diabetes mellitus constitutes a terminal condition. *Diseases of the Nervous System.*—Polyuria is a conspicuous symptom in certain cases of brain tumor, lesions of the medulla, and intracranial hemorrhage. It occurs also with some frequency in cerebral syphilis and has been observed in lesions of the cord. *Hysterical Polyuria.*—Copious, limpid urine of low specific gravity may simulate diabetes insipidus. The condition is transitory and the characteristic features of hysteria render the diagnosis a simple matter. *Contracted Kidney.*—There is frequently a large amount of urine of low specific gravity. Albumin is at times absent. It is, however, much more commonly present

in association with granular and hyaline casts. The signs of arteriosclerosis and cardiac hypertrophy are important. *Polyuria in Abdominal Diseases.*—This symptom is sometimes prominent in tuberculous peritonitis, aneurism of the abdominal aorta or iliac arteries, tumors of various kinds, and especially malignant new growths. *Hydronephrosis.*—The periodical discharge of large quantities of urine in connection with the subsidence of fluctuating abdominal tumor, which slowly reforms during the intervals, is of positive diagnostic significance. *Intercurrent Polyuria in Enteric Fever.*—I have reported a case of excessive urinary discharge, reaching a maximum of six litres in twenty-four hours, with slight increase of total urinary solids, occurring during the course of an otherwise mild attack of enteric fever, with disappearance of the polyuria upon convalescence. Other similar cases have been observed. *Malingering.*—Water may be added to the urine, but the absence of thirst, bulimia, and emaciation would lead to a suspicion of fraud which may be readily exposed upon investigation.

Prognosis.—The course of diabetes insipidus is extremely variable. It may be acute and rapid, or continue for many years with but slight deterioration of the general health. Recovery may occur and spontaneous cures have been noted. Death commonly results from some intercurrent affection.

IV. OBESITY.

Definition.—An excessive development of fat.

The condition is not always pathological. It is better to be fat and enjoy a normal amount of health and vigor than to reduce the fat by an unwise and rigorous regimen and depleting drugs, and become an invalid.

Etiology.—Predisposing Influences.—The hereditary tendency to obesity may be demonstrated in about fifty per cent. of the cases. This tendency may be manifest in childhood, in women after the first pregnancy, or not until middle life. It is more common in women than in men. Not alone in the hereditary cases, but also in those in which the tendency is acquired, is the condition more common in the female. There is a manifest relation between sexual inactivity and the tendency to corpulence. In males the tendency to accumulate excessive fat frequently begins in the fifth decade of life; in females at puberty, during the period of child-bearing, and at the grand climacteric. The distribution of fat varies at different periods of life. In infancy and childhood the undue accumulation is chiefly subcutaneous; in middle life it is visceral as well as subcutaneous, while in the aged the subcutaneous fat may disappear and that in the omentum, mesentery, pericardium, mediastinum, and around the kidneys persist. Persons of phlegmatic temperament, given to repose and the pleasures of the table, are more disposed to obesity than those who are sanguine, active, and self-denying.

The Actual Causes.—In general, obesity is due to the ingestion of excessive and improper food and an indolent and inactive life, but to this statement there are many exceptions. There are fat persons who are small eaters and exercise constant care in the selection of their diet, and among the obese are to be found men of superior intelligence and energy.

Fats, starches, and sugars taken in excess cause obesity. The habitual ingestion of large quantities of fluids and the abuse of alcohol are important etiological factors.

Symptoms.—Oertel describes a plethoric and an anæmic form. The first is more common in men who are high livers and consume much beer. The face is flushed, the subcutaneous and visceral fat are increased, but the muscular power is preserved for a long time. The second is especially encountered in chlorotic girls and anæmic women. The face is pallid, the skin white, the subcutaneous fat especially abundant, and the muscular power feeble. The ankles are often slightly œdematous. The hands and feet long remain free from disfiguring fat. Both types are too familiar to require detailed description.

Diagnosis.—Direct.—The recognition of obesity is a matter of little difficulty. The contour of the body and the disproportion between the height and weight of the individual are diagnostic. More difficulty arises in determining the line at which normal corpulence proper to the age, habits, and hereditary peculiarities of the individual ends, and obesity with its inconveniences and dangers begins. This can only be done by a careful study of individual cases.

Differential. — *Œdema.* — The irregular, doughy masses of subcutaneous fat, with the sharp folds of the skin and general distribution which characterize obesity, are in sharp contrast to the smooth, tense, glistening skin of anasarca, with its tendency to accumulate in the dependent tissues, where there is characteristic pitting upon pressure. *Myxœdema.* —The dense subcutaneous infiltration, symmetrical and of moderate extent, not pitting upon pressure, the implication of the hands, the pads, the mental state, and the prompt reaction to thyroid medication render the diagnosis clear. *Emphysema of the Subcutaneous Tissues.*—This rare condition may suggest obesity, but the history of the case, the circumscription of the swelling, and crackling upon palpation are characteristic.

Prognosis.—The outlook, varying with the causes, degree, symptoms, complications, and the disposition of the patient, ranges from favorable to positively ominous. It is less favorable in the hereditary than in the acquired form, in the anæmic than in the plethoric, and in the cases in which feeble action of the heart, arteriosclerosis, gout, albuminuria, or diabetes is present. Obese persons bear intercurrent febrile infections badly and usually make a tardy and unsatisfactory convalescence because of the slow regeneration of red blood-corpuscles and enfeebled recuperative powers.

Adiposis Tuberosa Simplex.—Under this term Anders has described a rare condition encountered in obese persons, characterized by the presence of circumscribed masses of fat in the subcutaneous tissues, particularly in the extremities and abdomen, and forming distinct, moderately dense, slightly movable, somewhat flattened tumors varying in size from a bean to a hen's egg and in number from six to twenty-four or more. These masses are not elevated above the surface and show no tendency to fuse together. They are sensitive to palpation and are sometimes, but not always, the seat of pain of variable intensity. The overlying skin is not adherent. Their etiology is not clear, but their relationship to corpulency

is manifest from the fact that they disappear as that condition is reduced under treatment. Adiposis tuberosa simplex is to be distinguished from: (1) *Adiposis dolorosa—Dercum's disease*—which is not amenable to treatment, and in which definite changes in the thyroid gland and the pituitary body, together with extensive interstitial neuritis and degeneration of the columns of Goll, have been found post mortem. (2) *Lipomata.*—Fatty tumors which are painless, soft and doughy, globular in shape, often lobulated, usually distinctly elevated above the surface, and which occur independently of general obesity and remain uninfluenced by treatment. (3) *Adenolipomatosis,* in which fatty accumulations develop in relation with the lymph-nodes of the neck, axillæ, or groins. These fat masses are symmetrical in distribution and occur in various chronic constitutional diseases, and only rarely are associated with general obesity. In fact, they may persist when, in consequence of the progress of the associated malady, emaciation has occurred or cachexia developed.

Adiposis Dolorosa.—Definition.—Dercum first called attention to "a disorder characterized by irregular, symmetrical deposits of fatty masses in various portions of the body, preceded by or attended with pain."

The disease occurs chiefly but not exclusively in women at middle life. Neuralgic pains precede and accompany the disorder. Irregular hyperæsthesia and paræsthesia occur. Fatty masses, sometimes of enormous size, lumpy, soft, and pendulous, form at various points of the body, in association with a general great increase of the subcutaneous fat. The face, hands, and feet are not affected. Atrophy of the thyroid body has been noted in some of the cases, and the administration of thyroid extract has been followed by relief of the neuralgia and diminution of the fat. Lesions of the pituitary body with interstitial neuritis and degeneration of the columns of Goll have also been found post mortem. The essential nature of the trouble is unknown. This disease differs from other forms of obesity in its unknown etiology, the distribution of the fat in masses, and the presence of marked nervous symptoms, especially pain.

V. AMYLOID DISEASE.

Lardaceous Disease; Waxy or Bacony Infiltration; Amyloidosis.

Definition. — A secondary affection in suppuration and syphilis, characterized by the formation and deposition of amyloid material or lardacein in the walls of the arteries and the viscera.

Pathologically, lardaceous disease is regarded as a degenerative change involving certain elements in the blood and an infiltration in the tissues of the organs. The process is general or constitutional. It affects no particular organ locally, but many organs and tissues at the same time, though not to the same degree. The organs commonly affected are, in the order of frequency, the kidney, the spleen, the liver, the intestines, the adrenals, and the lymph-glands. The pancreas, thyroids, testis, œsophagus, and endocardium are less frequently involved. The amyloid material is deposited at first in the arterioles, and in certain anatomical structures or regions as the intermediate or hepatic artery zone of the liver lobule, the

Malpighian tufts and the cortex generally in the kidney, the Malpighian bodies in the spleen, and the arterioles in the mucous membranes. In many of the cases the material is distributed throughout the whole of the organ, with the result that the solid viscera are increased in bulk, sometimes to an enormous extent. This increase is sometimes, especially in the kidney, followed by contraction.

Etiology.—Suppuration, chronic or recent, with or without discharge, is present in the great majority of the cases. Pulmonary tuberculosis and disease of bone are the most frequent causes of suppuration antecedent to amyloid disease. Tuberculosis without suppuration does not appear to be a factor. On the other hand, suppuration, in the absence of tuberculosis or other specific constitutional infection, is a very common antecedent. Syphilis without purulent lesions must be recognized as a cause. Malaria is a possible cause, but its agency is still in question. As a rule, the suppuration has been prolonged, but there are exceptions to this rule. The amyloid process develops during the suppurative process, but may not cause recognizable clinical manifestations until after suppuration has continued for years, or not until after it has ceased. Males are more liable than females, not because of any differences incident to sex, but because they are more exposed to injuries and diseases attended by purulent lesions, and to syphilis. The predisposition associated with age is shown by the rarity of amyloid disease before ten and after fifty. It is most common between twenty and forty.

Symptoms.—There are general manifestations of amyloid disease irrespective of the visceral changes. The suppurative primary diseases have almost always produced changes that are characteristic or at least suggestive. These are manifest in the signs of advanced phthisis, the deformities of old empyema or bone disease, especially those forms which involve the spine and joints. Since such processes are attended with wasting, the pinched features, emaciated frame, and shrunken extremities are highly suggestive, especially as they are associated with a prominent or enormously distended abdomen due to the overgrown size of the amyloid viscera. A muddy pallor of the skin, dropsical effusions in the dependent parts, diarrhœa, polyuria, thirst, albuminuria, and great weakness complete the picture. The onset is insidious.

It is customary to describe the clinical manifestations of amyloid disease in the organs in connection with the various diseases of each; it seems, however, more appropriate and more useful for the purposes of the diagnostician to consider them here.

1. **Amyloid Kidney.**—The process is associated with wide-spread amyloid degeneration in other viscera due to suppurative diseases or syphilis. It has been attributed also in some instances to leukæmia, chronic lead intoxication, and gout. It is frequently associated with the chronic form of parenchymatous nephritis. The kidney is usually much increased in size; in exceptional cases it does not exceed the normal kidney in this respect. The surface is smooth and the stellate veins are conspicuous. The organ is firm. Upon section the cortex is thickened, the glomeruli distinct, and the pyramids of a deep red color. The iodine test shows a deep mahogany color most marked in the Malpighian tufts and straight vessels.

Symptoms.—There are urinary features of importance. The quantity is increased, the color pale and transparent, the specific gravity low. Albumin is, as a rule, abundant; exceptionally there is a mere trace or it may be absent. Hyaline, fatty, and granular tube-casts are present, and occasionally the amyloid color-reaction may be obtained. Dropsy is usually present, but there are cases in which it does not occur. Diarrhœa is common. Increased arterial tension, cardiac hypertrophy, retinal lesions, and uræmia do not occur except in cases of amyloid degeneration affecting the small granular kidney.

Diagnosis.—The renal symptoms alone have little diagnostic value. Their development in connection with prolonged suppuration or syphilis, and in association with an enlarged liver and spleen, and persistent diarrhœa, is highly suggestive.

2. **Amyloid Liver.**—The etiological relations of this condition are the same. It constitutes an important visceral manifestation of amyloid disease. The organ is large and may attain an enormous size. It is firm, dense, and resistant. Upon section the surface is pale and presents at the edges a slightly translucent appearance. It responds to the iodine test by the development of a mahogany-brown color in the affected areas. The capsule is smooth and the borders of the enlarged organ are rounded and blunt. Exceptionally the margins are sharp and well-defined. The enlargement is commonly uniform

Symptoms.—There are no characteristic hepatic features. Jaundice does not occur. The stools are sometimes light but not clay-colored. There are no signs of portal obstruction. The spleen is often enlarged.

Diagnosis.—The history taken in connection with progressive enlargement of the liver which, upon palpation, yields the above signs, together with enlarged spleen, polyuria with or without albumin, and diarrhœa, constitutes positive evidence of the presence of amyloid disease.

3. **Amyloid Spleen.**—The organ is not usually greatly enlarged but can be readily recognized upon palpation. Its edges are thick and rounded and its consistence dense. Upon section the lardaceous infiltration is seen to affect especially the Malpighian bodies, which are prominent and glistening, giving rise to the appearance described as "sago spleen." In some cases the intervening tissue is more or less diffusely affected.

There are no special symptoms.

Diagnosis.—A history of suppuration or syphilis, a cachectic state, emaciation, prominent abdomen due to coincident enlargement of the liver, urinary changes, and diarrhœa when present justify the assumption that an enlarged spleen is amyloid.

4. **Amyloid Disease of the Intestines.**—The blood-vessels of the entire digestive tract may be affected. More commonly the small intestine, especially the ileum, or the colon is the seat of the disease.

Symptoms.—When slight in intensity or of limited extent the disease presents no features by which it can be recognized clinically. The one symptom of advanced or extensive amyloid degeneration in the intestines is persistent diarrhœa. The stools are variable in consistency and number. They are usually thin and liquid but without distinctive characters. Especially are they not bloody. They are not attended by colic or tenesmus and the abdomen is not sensitive to pressure.

Diagnosis.—The recognition of the disease is difficult and uncertain, since diarrhœa without pain and tenderness and equally intractable may occur in various other intestinal disease. The association of this symptom with the above-described clinical manifestations in a person suffering, or who has suffered, from prolonged suppuration, or who has syphilis, renders it in the highest degree probable that there is amyloid disease of the gut.

Prognosis in Amyloid Disease.—The outlook is doubly unfavorable. The antecedent disease is a frequent, the amyloid disease a common, cause of death. The highest mortality among the visceral forms relates to the kidneys, the next to the intestines. Extensive lardaceous degeneration of the liver and spleen may occur without special symptoms of importance, and without great impairment of health in addition to that caused by the primary disease.

X.

THE DIAGNOSIS OF DISEASES DUE TO THE DEFICIENCY OR ABSENCE OF CERTAIN NORMAL ELEMENTS OF FOOD: DEFICIENCY DISEASES.

"The term vitamines is used to designate the group of substances in the animal diet which are present in minute traces, whose nature and chemical constitution are unknown, and whose absence will result in a series of pathological changes which can only be overcome by addition of these essential factors." (A. B. Macallum.)

"When vitamines are taken into the body their action is to stimulate the internal secretions. By setting free these secretions they act as hormones. These hormones again stimulate the enzymes." (Louis Fischer.)

Funk using dried yeast and rice polishings and experimenting with pigeons placed the deficiency hypothesis upon a scientific basis (1913). This observer states that potatoes, many cereals, meats that have been subjected to great heat, and milk that has been boiled do not contain sufficient vitamines to prevent the development of polyneuritis. Much work is being done both in the laboratory and the clinic in this field of investigation, especially as regards the artificial feeding of infants, the inordinate use of canned foods and monotonous and unbalanced diets in general.

I. BERI-BERI.

Kakke.

Definition.—An endemic and epidemic disease resulting from a prolonged diet deficient in a neuritis-preventing vitamine, widely prevalent in tropical and subtropical countries of the East, and characterized by multiple neuritis causing either atrophy, paralysis, or general œdema, or both.

Etiology.—Predisposing Influences.—This disease is very common in China, Japan, the Philippines, and the Malay Archipelago. Local

epidemics have been observed in Australia. It is prevalent in Brazil and occurs in the West Indies. Imported cases are not uncommon in the seaport cities of the United States, especially on the Pacific Coast. In a few instances American fishermen have suffered from it, both upon the Grand Banks and in shore fishing. In its prevalence as determined by season and locality it is not unlike malaria, being most common in the hot and rainy seasons. Absence of sunlight and air, and overcrowding are important favoring conditions, hence beri-beri is frequent in jails and asylums. It is also a common and troublesome disease on shipboard, both in naval and commercial service. It is especially a disease of fishermen, a fact erroneously attributed to exposure and wet. In districts where the disease is endemic, and even in epidemics, the native races chiefly suffer. The imported coolies are especially liable to it. Young men from 16 to 30 are most frequently attacked, but no period of life is exempt. Males suffer much more often than females.

Exciting Cause.—Beri-beri was long regarded as a food disease and attributed to rice which has undergone certain unknown changes, or fish

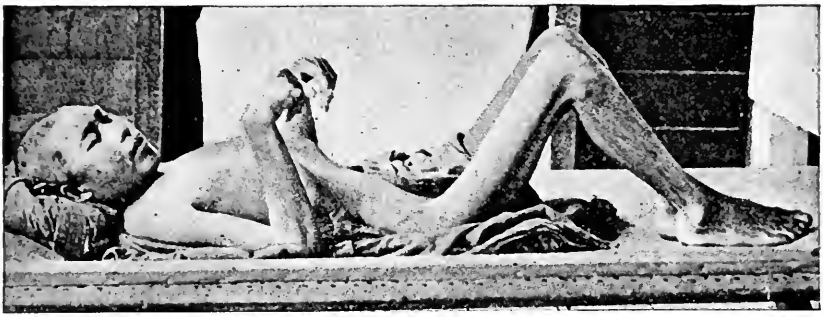


Fig. 311.—Atrophic variety of beri-beri showing muscular atrophy and wrist drop.—(*Journal of Tropical Medicine.*)—*International Clinics.*

eaten raw or improperly cooked. The Dutch physicians, who have first-hand knowledge of the disease upon an extensive scale, regarded beri-beri as an infectious disease. Recent observations conducted by the Board for the Study of Tropical Diseases in the Philippine Islands have shown that beri-beri is a food disease dependent upon an excess of polished rice—*i.e.*, rice from which the pericarp has been wholly removed—and that (a) a reduction in the rice ration with an increase in the legumen and (b) the substitution of under milled rice—*i.e.*, rice in which some part of the pericarp remains upon the grain—were followed by the disappearance of beri-beri from the native scout organization during the last half of the year 1910. This body is 5,000 strong, and no other cause could be shown to be operative, nor was there any corresponding decrease in the incidence of beri-beri in the general Filipino population or in the native constabulary. Heiser, in an outbreak of beri-beri in the leper colony at Culion in the Philippine Islands (1911–1912) found that the outbreak ceased when unpolished rice was substituted for the polished rice previously largely constituting the diet.

Many similar observations have been reported and the general use

of unpolished rice has been followed by a marked diminution of the prevalence of beri-beri in rice eating countries.

The dietary fault is not in the presence of some toxic principle but in the loss of the vitamine in the pericarp, which has a profound influence upon nutrition and in particular in preventing neuritis.

Symptoms.—The period required for the disease to develop after the adoption of the diet lacking in vitamine appears to be of long duration, 60–160 days. A dry and a wet form were once recognized. The following clinical forms are described but they are not sharply differentiated: 1. Undeveloped or Larval Form.—The onset is marked with catarrhal phenomena which are followed by nervous symptoms, such as modifications of sensations, paræsthesias, hyperæsthesia and pain, and loss of power in the limbs, chiefly in the legs and feet. The muscles are not only weak, they are also tender upon pressure. There is in many cases even in this form œdema of the feet and ankles, but it is slight. Cardiac palpitation, dyspnœa, and abdominal distress may be present. This symptom-complex is of variable duration. After a week or two, or several months, recovery takes place, but there is a tendency to recurrence. 2. Atrophic Forms; so-called Dry Form.—Early symptoms are the same as in the incomplete form. There is, however, more pain; wasting of the muscles sets in and progresses rapidly so that in a short time paralytic phenomena are pronounced and the power of movement is greatly impaired. Cardiac derangements and dropsy are not prominent. 3. Dropsical, or so-called Wet Form.—With similar symptoms œdema occurs early and soon becomes general with effusions into the serous sacs. Muscular and neural symptoms are less pronounced than in the atrophic forms and the wasting is not apparent until after the anasarca subsides. 4. Acute Cardiac, or Pernicious Form.—The symptoms which characterize the rudimentary form are followed by the evidences of acute cardiac asthenia, which may persist for several weeks or terminate fatally in the course of a day or two.

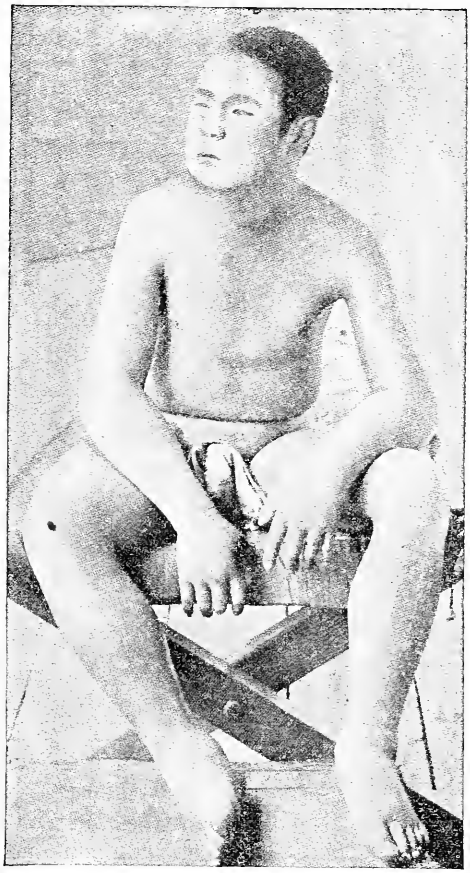


Fig. 312.—Œdematous variety of beri-beri.—(*Journal of Tropical Medicine.*)—*International Clinics.*

Diagnosis.—Direct.—In countries in which beri-beri is endemic there is little difficulty in the diagnosis.

Differential.—Only in an isolated imported case could multiple neuritis from other causes, as alcohol, arsenic, etc., occasion difficulty. Here the anamnesis is most important and dropsical symptoms most suggestive.

Prognosis.—The disease is essentially subacute and chronic and lasts a varying time, measured by days or months. The acute cardiac forms usually become chronic after a number of days or weeks. Recurrences are common, especially in the wet season. The mortality varies from 2 or 3 to 50 per cent., and is very high among coolie laborers imported into a beri-beri district.

II. SCURVY.

Scorbutus.

Definition.—A nutritional disease, due mainly to improper food, and characterized by anæmia, great debility, swollen and ulcerated gums, and subcutaneous, submucous, and subperiosteal hemorrhages.

Etiology.—Predisposing Influences.—Scurvy is a disease of unsanitary conditions and has occurred in all parts of the world in which such conditions prevail. It has been known from the earliest historical periods and has been the scourge of armies in the field, soldiers and sailors on long voyages, and the peoples of beleaguered cities. This disease may occur in any climate, but is more common in northern climates and cold countries. It is more frequent in cold weather than in warm, in rigorous winters than in mild, and in periods of famine. Among adults males suffer more frequently than females, and more severely. In besieged cities where the conditions affecting the sexes have been very similar, the proportion of men affected has exceeded that of women. It is most common in adult life, but affects children and the aged. A special form is known as *infantile scurvy*. Malaria, syphilis, dysentery, alcoholism, defective teeth, and gastro-intestinal disorders constitute predisposing influences of recognized importance. Anxiety, prolonged fear, nervous depression, and nostalgia play an important part in the predisposition to the disease, but are incapable of causing it.

Pathogenesis.—There are five hypotheses as to the essential cause of the disease:

1. That it is due to the absence in the food of certain substances present in fresh vegetables. The nature of these substances has not been positively determined. They have been thought to be various organic salts present in fruits and vegetables; or the potassium salts (Garrod). According to Ralfe the lacking substances are the malates, citrates, and lactates, from which are derived the carbonates upon which the alkalinity of the blood depends. This view appears to be disproved by the fact that scurvy does not prevail among the Esquimaux, who live exclusively upon animal foods and fats which are often tainted, and that of Nansen and other Arctic explorers who, living for months upon a similar diet in most unhygienic surroundings, escaped the disease.

2. That it is due to toxic substances of unknown character, products of decomposition, in the food.

3. That it is essentially a toxæmia, resulting from the absorption of poisonous substances from the gastro-intestinal tract, produced by micro-organisms in the intestinal contents.

4. That it is an infection depending upon an unknown specific micro-organism, for which depressing influences, unsanitary conditions, and improper food prepare the soil.

5. Recent investigations point to the probability that scurvy may be due to the absence of one of the group of vitamines.

It is to be noted that prolonged insufficiency of food—starvation—on the one hand does not necessarily result in scurvy, and that, upon the other, the disease may occur with an abundant diet of improper and monotonous food.

Symptoms.—Scurvy occurs as an epidemic, endemic, and sporadic disease. Sporadic cases are often encountered in prisons, almshouses, hospitals, and other similar institutions, and occasionally among well-to-do persons in private life, who, as a matter of fancy, or for some other reason, have lived for a long period upon a restricted and unvaried diet. In the United States, where scurvy is at present a rare disease, these sporadic cases are often unrecognized. The disease is insidious in its development. It may be subacute or chronic. In very rare cases the onset is abrupt. The course in ordinary cases is progressive and attended by fluctuations in the intensity of the symptoms. The early manifestations consist of weakness, pallor, and loss of weight. The gums become swollen, spongy, and ulcerated. Sometimes they show the continuous oozing of blood. The teeth are loosened and frequently fall out. The tongue is enlarged, red, and frequently heavily coated. Submucous hemorrhages in the mouth occur and the breath is disgustingly fetid. The skin becomes dry and harsh and petechiæ appear in and around the hair-follicles, at first upon the legs and later upon the arms and trunk. Subperiosteal extravasations upon the legs often give rise to painful nodes, which sometimes break down and form deep ulcerations. Subcutaneous hemorrhages arise at points of injury or pressure, and brawny indurations occur in the subcutaneous tissues, with discolorations of the overlying skin. Epistaxis is common and free bleeding from other mucous surfaces takes place in the graver cases. Hemorrhagic infarcts in the lungs and spleen with characteristic symptoms may be noted. Feeble action of the heart with arrhythmia and palpitation are frequent and there is often a basic systolic murmur. There is anorexia and inability to masticate food owing to the condition of the gums. Constipation is the rule. Arthritis has been noted. The urine is albuminous. Urea is diminished. The other constituents show inconstant changes. Mental depression and languor are common. Delirium and coma occur as terminal events. Subconjunctival and intra-ocular hemorrhages are of common occurrence and hemeralopia and nyctalopia are occasional symptoms. Fever is not usual, but pyrexia of irregular type may occur in the presence of complications, such as pleurisy, pericarditis, or abscess formation.

There is secondary anæmia with neutrophilic leucocytosis. Nucleated red corpuscles are often present. The color index is low.

Complications and Sequels.—Abscesses, inflammation of the serous sacs with hemorrhagic effusion, croupous pneumonia and bronchopneumonia, pulmonary gangrene, and pulmonary œdema as terminal events constitute the most serious complications. Gastro-intestinal complications are common. Ankylosis of joints that have been inflamed, particularly the elbow, knee, and ankle, may cause permanent deformity.

Diagnosis.—The DIRECT DIAGNOSIS in the epidemic and endemic disease is unattended with difficulty. The surroundings and circumstances of the patient, the condition of the gums, the petechiæ and ecchymoses, the langnor and anæmia, the nodes due to subperiosteal bleeding, and the results of dietetic and hygienic treatment establish the nature of the disease.

DIFFERENTIAL DIAGNOSIS.—The diagnosis in sporadic cases may be difficult, especially in persons living in affluence in whom errors of diet and unhygienic surroundings are unsuspected. The foregoing diagnostic criteria are important. The lesions of the gums are absent in early infancy prior to dentition, and in aged persons who have lost their teeth. Pericarditis and pleural effusion of scorbutic origin may, in the absence of the lesions of the gums and subcutaneous hemorrhages, give rise to serious diagnostic difficulty. The anamnesis, the evidences of deep-seated hemorrhages, and the results of treatment are important. *Purpura.*—The various forms of purpura differ from scurvy in their causal relations, the absence of the peculiar lesions of the gums, and the absence of the deeper-seated hemorrhages.

THERAPEUTIC DIAGNOSIS.—An abundance of fresh vegetables and meat, such as constitutes an ordinary wholesome mixed diet, is usually followed by a remarkable disappearance of the symptoms, even in cases of great severity. The articles of diet which have the reputation of being especially valuable comprise potatoes, lettuce, cabbage, spinach, and fresh fruits and fruit juices, as lemon and lime juice. From the time that the regulations of the Board of Trade have required that a sufficient quantity of such articles be included in ships' supplies, the occurrence of scurvy among sailors has become a rare event.

Prognosis.—The cases are apt to recover unless the causal conditions persist or they are far advanced when treatment is begun. This disease is now infrequent and the mortality low. Death results from progressive inanition, sudden syncope, large serous effusions, pneumonia, pulmonary œdema, meningeal hemorrhage, or sepsis.

III. INFANTILE SCURVY.

Barlow's Disease.

Definition.—A nutritional disease of young infants, due to improper food and characterized by subperiosteal hemorrhages, particularly in the lower extremities, a form of pseudoparalysis, and a cachectic condition. Our knowledge of this disease, which was formerly confounded with rickets and infantile syphilis, is of comparatively recent origin (1881-1883).

Etiology. — Predisposing Influences.—Infantile scurvy is more common among the well-to-do than among the poor, a condition to be explained, first, by the fact that the children of the former are more commonly fed upon artificial foods, and second, that among the latter the child has at an early age a more varied diet, into which enter to some extent articles of ordinary table food quite unknown to children of the same period among the affluent. Scurvy begins most commonly between the sixth and eighteenth months. It is rare earlier, but has been observed as late as the fifth year. Rheumatism, syphilis, and rickets have been supposed to bear a causal relation to infantile scurvy. Of these the first two have nothing to do with the disease in question and the last, though sometimes associated with it, is wholly different in its etiology, pathology, and symptomatology, and when present in the same individual persists when the scorbutic symptoms have disappeared

The Immediate Cause.—Improper diet is the cause of this disease. The various commercial foods, including condensed milk (especially when prepared with water), sterilized milk, and other artificial foods (particularly when administered in unvarying monotony), are found to have constituted the diet in almost every case for a considerable period prior to the manifestation of the symptoms. There is good reason to believe that the fault in diet consists in the absence of vitamines.

Symptoms.—The disease shows itself as an insidiously developing cachexia. The child is fretful and peevish. It lies quiet when undisturbed, with its thighs and legs strongly flexed, but screams when any attempt is made to extend them. Obscure swellings due to subperiosteal hemorrhages may be observed on both lower extremities but they are not symmetrical. These ill-defined, tumor-like prominences occupy the lower ends of the femurs, the tibiæ, and less frequently the bones of the forearms. They are most marked just above the epiphyseal junction and extend along the shafts of the bones. Similar swellings may sometimes be found upon the scapulæ. The overlying tissues are boggy and slightly œdematous and the skin is somewhat tense. As the disease progresses the whole limb becomes thickened. Presently the limbs assume a different position, being no longer drawn up, but everted and motionless—*pseudoparalysis*. The joints are not involved. Separation of the epiphyses and fracture may occur in severe cases, these lesions being manifest by crepitus and further deformity. Barlow described a remarkable depression of the sternum and costal cartilages. Proptosis of the eyeballs, more marked upon one side than the other, with œdema and slight discoloration of the eyelids, may occur in advanced cases. Petechiæ occur, but are much less conspicuous than in the scurvy of adults, but hemorrhages from the mucous surfaces are common. Anæmia—3,000,000 to 2,000,000 or lower—is proportionate to the severity of the case. The white corpuscles show no constant changes. The color of the skin is pallid and earthy: emaciation is not a marked feature: asthenia is extreme. The temperature may be normal or slightly subnormal, with occasional transient rises to 102° or more, these usually accompanying the signs of fresh subperiosteal hemorrhages. If the teeth have appeared the gums may be swollen and spongy.

Diagnosis.—Direct.—It is a matter of surprise that the true character of infantile scurvy is so often overlooked. Few diseases of infants present a more characteristic symptom-complex or a more obvious etiology. The attitude, the behavior of the child upon being handled, the anæmia, the elongated, subperiosteal nodes and thickening of the limbs, the immobile, forced flexion of the limbs in the early, and the pseudoparalysis in the later, course of the affection are diagnostic. Proptosis and œdema of the eyeballs are significant.

Differential.—*Rickets.*—The gastro-intestinal symptoms are more prominent than in scurvy. The rachitic rosary and the lesions of the bones are characteristic. The boggy swellings, protrusion of the eyeballs, petechiæ, and spongy gums when the teeth are present do not occur in rickets. The two diseases may, however, be associated. The forms of *purpura* resemble scurvy only in the presence of petechial and other hemorrhages, but the distribution of these lesions and the absence of all else characteristic of the latter disease render the diagnosis an easy matter. *Infantile Paralysis.*—The pseudoparalysis may suggest this affection, but the history of the case, the sudden onset, the absence of pain, tenderness, and the localized swellings are diagnostic. *Syphilitic Pseudoparalysis: Parrot's Disease.*—Sudden loss of motion in the lower or upper limbs, or both, with great pain on passive movement, and crepitus due to a separation of the epiphyses may present a superficial resemblance to infantile scurvy. This resemblance ceases upon a proper consideration of the anamnesis and the lesions. The diagnostic criteria of congenital syphilis are usually unmistakable. In any doubtful case an etiological diagnosis based upon the nature of the diet will be helpful.

Prognosis.—The outlook is favorable in cases early recognized. Complete recovery, with the disappearance of the lesions, often takes place in the course of two to four weeks after the institution of a proper diet. More advanced cases recover more slowly.

IV. PELLAGRA.

Definition.—A chronic disease of undetermined causation, characterized by a peculiar dermatitis of definite distribution, marked gastro-intestinal derangements, great physical and mental depression, profound disturbances of nutrition and terminal dementia. Its progress is at first periodic, later continuous. Its occurrence may be sporadic, endemic or epidemic. It is not directly transmissible from the sick to the well.

Etiology.—Predisposing Influences.—The geographical distribution is largely restricted to northern Italy, Spain and the south of France. Cases have been observed in Egypt. Recent observations have shown it to be widely prevalent in the United States, especially in institutions for the care of the insane. Its prevalence is neither coterminous with nor restricted to maize-consuming districts. It is a disease of the poor and of rural populations. It occurs chiefly in middle life, but cases have been observed at every age. Women suffer more frequently than men.

Exciting Cause.—Two hypotheses are defended: (1) That of Lom-

broso, who maintains that pellagra is caused by eating bad maize and is due to some poisonous principle associated with maize—an opinion until recently universally accepted; (2) That of Sambon, who holds that the presence of pellagra is closely associated with streams of running water and the presence of a suctorial fly of the genus *Limulium,* which abounds in certain districts bordering upon the streams.

The failure of these views has lead to the suggestion of a third working hypothesis, "that places pellagra among the diseases due to some special food deficiency, probably a deficiency in some vitamine" (Barker).

Symptoms.—The early symptoms are not distinctive. They consist of weakness, sleeplessness, headache, vertigo, dyspepsia, muscular cramps and pains referred to the spine and joints. These attacks occur in the spring or summer and subside in the autumn, only to recur again in the following spring. Fever is not common except in the severer cases, in which irregular pyrexia—102°–105° F. (39.0°–40.5° C.) is frequently observed. The eruption is symmetrical and affects chiefly the parts exposed to the sun, as the hands, wrist, neck and upper part of the chest and the feet and legs. It has the characters of an intense erythema with petechiæ and in severe cases bullæ, which rupture and leave indolent ulcers. Upon its subsidence the skin is left thickened and deeply pigmented. The dermatitis recurs annually, but after the fourth or fifth year the integument involved undergoes atrophic changes. The nails and hair are not affected. After some years the symptoms become continuous. Dyspepsia and diarrhœa are pronounced. Chronic stomatitis characterized by salivation and a cardinal red or bluish black stippled tongue, often denuded of epithelium, is common. Wasting, weakness and mental depression progressively increase. Vertigo, unilateral or bilateral mydriasis, twitchings, tremors, epileptiform seizures of the cortical type and palsies occur. The reflexes are increased; ankle-clonus is often present. The gait is uncertain but not ataxic. Definite derangements of sensation do not occur. The later stages of the disease are characterized by melancholia, mania and dementia. At this period there are forms of palsy with diminished or absent reflexes, the signs of a peripheral neuritis. A majority of the cases in the United States have been discovered in asylums for the insane poor, but recent observations and a growing knowledge of the facts among practitioners render it probable that many obscure cases of ill health among the country people of the South and Southwestern States are pellagrous.

Diagnosis.—The direct diagnosis rests upon the foregoing periodical and progressive phenomena and may be made without difficulty. The resemblance to general paresis and leprosy is of the most superficial character.

Prognosis.—In pellagra the prognosis is uncertain. Mild cases may abruptly become severe. The gravity of the case cannot be gauged by the cutaneous lesions. The mortality in the United States ranges between 25 and 40 per cent. It is much lower in Italy. After the third or fourth annual periodic recurrence, especially when mental symptoms have shown themselves, the outlook is very unfavorable. Death occurs from progressive wasting and asthenia or from intercurrent disease.

V. RICKETS

Rhachitis.

Definition.—A disease of infants due to improper diet and characterized by impaired nutrition of the tissues of the body and specific alterations of the skeleton.

Etiology.—Predisposing Influences.—All those conditions which involve neglect of hygiene, and especially of alimentary hygiene, favor the development of rickets. The geographical distribution of this disease is wide. It abounds in great cities and crowded industrial centres. It is more common in Europe than America. In this country it is especially prevalent among the children of recent immigrants. The great frequency of rickets among the children in the Italian and negro colonies of American cities is due not to racial but to social conditions. Rickets affects male and female children to the same extent and degree. If we except the rare condition known as fetal rickets—*achondroplasia, chondrodystrophia fœtalis*—and the late form described by Jenner—the *osteomalacia of puberty*—rickets is a disease of the first two years of life, a period corresponding to the first dentition. It rarely begins before the sixth month or after the third year. Rickets is especially a disease of poverty and all that poverty entails—want of sunlight and want of fresh air, neglect, filth, and insufficient and improper food. Rickets, like scurvy, occasionally occurs as a sporadic disease in the families of the well-to-do. Rickets has been looked upon as a manifestation of congenital syphilis, but this view has been abandoned alike upon etiological and pathological grounds. A syphilitic child is not rhachitic, though it may acquire rickets, and the two conditions frequently coexist. There is no evidence that rickets is hereditary. The endemic and epidemic prevalence of the disease, under certain social conditions, is neither evidence of its hereditary nature nor of its contagiousness, as has been assumed.

The Cause.—An improper diet is the essential cause of the disease. Prolonged lactation and suckling the child after pregnancy has occurred bring the milk of the nursing mother within the category of improper food. Cow's milk, foods rich in starches, condensed milk, and the various commercial infant's foods are responsible for a large proportion of the cases. Deficiency in fat and proteids, and failure in the assimilation of the lime salts, constitute the chief alimentary defects.

The Pathogenesis.—The essential pathological fault is obscure. There are two hypotheses: (a) a primary defect in the tissue of the bones which interferes with their appropriating salts from the blood; and (b) an abnormal loss of calcium by way of the bowel. It is worthy of consideration that the dietary fault may be the absence of some principle that controls the selective relation of bone tissue for calcium salts—vitamine.

Symptoms.—Rickets is a chronic disease of insidious onset, beginning during the first dentition and usually before the child begins to walk. It is preceded by digestive disorders of varying degree, and impaired nutrition, but not necessarily by emaciation. The child is often pallid, plump, and

soft. Slight fever, irritability, and poor sleep are suggestive symptoms. He is feeble and unsteady on his feet and disinclined to walk. There is diffuse tenderness of the tissues and unwillingness to be handled or touched. Free sweating, especially about the head and neck, is common. The weakness of the muscles, and especially in the legs, is suggestive of partial paralysis—*pseudoparalysis* of rickets. The skeletal changes appear early in the course of the disease and are characteristic. They consist of: 1. The "RHACHITIC ROSARY," composed of nodular enlargements of the ribs at their juncture with the cartilages on both sides. These enlargements may be readily felt upon palpation and in thin children may be recognized upon inspection. They appear early and gradually increase in size until some time in the second year, after which they gradually disappear. 2. CHANGES IN THE THORAX.—Shallow furrows, corresponding to the junction of the cartilages with the ribs, pass obliquely downward and outward. A similar transverse depression extends from the level of the ensiform

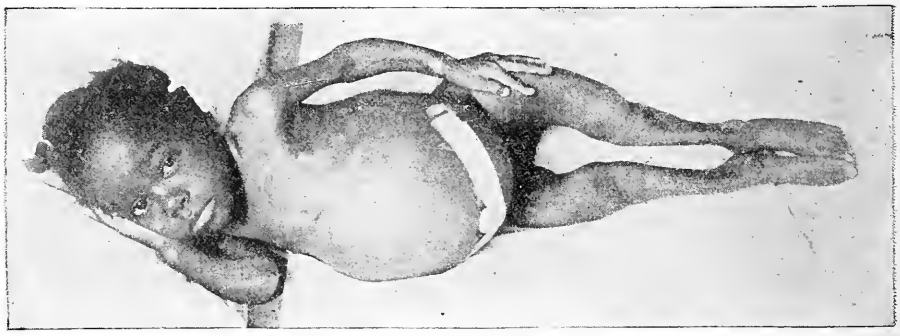


FIG. 314.—Rickets. Showing the epiphyseal enlargements, the rosary, the distended belly, and the deformities as a whole.—Pennsylvania Hospital.

cartilage toward the infra-axillary space—*Harrison's groove.* The sternum projects, particularly in its lower half, giving rise to the prominent deformity known as *chicken-* or *pigeon-breast.* These changes in the contour of the chest are not peculiar to rickets and may occur in any condition habitually interfering with inspiration in early life. 3. CHANGES IN THE HEAD.—As a rule the head appears large, the frontal and parietal eminences are exaggerated, and the fontanelles remain open for a long time. The forehead is prominent, the top of the skull flattened, and, in some cases, the head viewed from above appears square—*caput quadratum. Craniotabes.*—There are circumscribed areas, mostly in the occipital, parietal, and squamous portions of the temporal bones, in which, in consequence of decalcification, the skull may yield to the pressure of the finger, giving rise to "*parchment crackling.*" This condition has been observed also in syphilis. A systolic murmur may frequently be heard over the anterior fontanelle or in the temporal region. This auscultatory phenomenon is sometimes heard in healthy children. The bones of the face also show changes, especially in the maxillæ, which are small and angular. The normal course of dentition is deranged and retarded. The first teeth may not appear until

some time in the second year and undergo caries at the time of their eruption. The cephalic changes of rickets are very often first in the point of time. 4. Changes in the Pelvis.—The changes in the pelvis are of especial importance in female children, since they lead to deformities with narrowing, which interfere with natural labor and frequently render it impossible. 5. Changes in the Extremities.—The scapulæ are not usually affected. The clavicles are often thickened at the sternal ends and at the point of insertion of the sternocleido muscles; their curves are exaggerated and they are shortened. The rhachitic deformities are most conspicuous in the long bones. They consist of enlargements in the region of the junction of the shaft and epiphysis, and curvatures, which in the lower extremities cause a corresponding diminution in the height of the individual. The enlargements in the upper extremities are most marked at the distal ends of the radius and ulna. To a less degree the lower end of the humerus may be affected. *The Rhachitic Hand.*—Koplik has described a deformity of the hands which occurs in rickets, consisting in thickening and bowing of the phalanges of the fingers, associated with laxity of the ligamentous structures of the phalangeal joints. The changes give rise to a somewhat characteristic appearance of elongation of the fingers and plumpness of the whole hand. They have been observed in connection with the ordinary lesions and deformities of well-marked rickets. In cases attended by pains in the bones "the rhachitic hand" may suggest syphilis, but the association of the lesions of infantile syphilis render the differential diagnosis easy. In the lower extremities the lower end of the tibia, of the fibula, and of the femur show progressive enlargements proportionate to the severity of the case. If the child walks, the femurs are curved forward and the bones of the legs forward and outward. Exceptionally the curves may cause the deformity known as knock-knee. These abnormal curvatures are due to the muscular traction and the weight of the body upon the decalcified and softened bone.

Fig. 315.—The skeleton of the body shown in Fig. 314.

The liver is enlarged: the spleen enlarged and palpable. There is usually more or less flatulent distention. These conditions combine to render the belly large and protuberant, a condition made more conspicuous by the relatively small size of the thorax.

The urine shows no constant changes. There is slight anæmia; the

hæmoglobin is decreased; leucocytosis may or may not be present. The nervous symptoms increase with the severity of the other symptoms. Convulsions are common. Tetany and laryngismus stridulus are occasional intercurrent affections. The growth of the child is greatly retarded and many dwarfs are rhachitic.

Diagnosis.—Direct.—Many cases are so slight as to escape recognition. Weakness, fretfulness, pallor, diffuse soreness, profuse sweating of the head during sleep, an open fontanelle, and irregular evening fever justify a provisional diagnosis, especially when defects of hygiene and diet exist. When to these symptoms are added the skeletal changes above described, especially those which first appear, namely, the rosary and craniotabes, it becomes positive.

Prognosis.—The slighter forms are amenable to treatment and recovery takes place without deformity. The graver cases recover more slowly and with lasting skeletal changes. The disease is essentially chronic, and, though not in itself fatal, renders the patient peculiarly liable to intercurrent affections, while it at the same time diminishes the powers of resistance.

XI.

THE DIAGNOSIS OF THE DISEASES OF THE DIGESTIVE SYSTEM.

(DISEASES OF THE MOUTH, TONGUE, GUMS, SALIVARY GLANDS, PHARYNX, TONSILS, AND ŒSOPHAGUS ARE CONSIDERED IN VOL. I.)

I. DISEASES OF THE STOMACH.

i. Acute Gastritis.

The following forms are recognized: toxic, phlegmonous, diphtheritic, parasitic, and dietetic.

1. **Toxic Gastritis.**—An intense form of inflammation produced by various irritant and corrosive poisons. It varies in degree according to the nature, concentration, and quantity of the poison, and the length of time it has remained in contact with the gastric mucosa.

Symptoms.—Sudden pain, nausea, retching, and vomiting occur. The vomitus in severe cases consists of blood-stained food remnants, mucus, shreds of mucous membrane, and the poison itself. Thirst and dysphagia are distressing. Later there is usually diarrhœa. Collapse comes on rapidly: the temperature, at first subnormal, rises later; and jaundice is not uncommon. Epigastric and abdominal tenderness may be followed by the signs of general peritonitis. Death occurs from the intoxication, or from exhaustion, convulsions, or suffocation. When recovery takes place, ulceration with stenoses or chronic gastritis supervene. The direct diagnosis depends upon the anamnesis, the evidences of the corroding poison upon the lips, mouth, and pharynx, the odor of the breath in certain cases, the presence of the vial or package which contained the poison, the analysis of the vomitus, the analysis of the urine, and the foregoing associated symptoms. The prognosis is, in the main, unfavorable.

It depends, however, upon the nature of the poison, its amount, the time elapsing before its removal or the administration of antidotes, the direct damage to the stomach itself, the intensity of the collapse, and the occurrence of peritonitis.

2. **Phlegmonous Gastritis.**—Acute diffuse or circumscribed suppurative inflammation of the gastric submucosa.

Etiology.—This is a very rare affection. The cases are primary, in which alcoholism appears to be a predisposing influence, and trauma, faulty diet, and various irritant poisons the exciting causes; and secondary, in which the various general febrile infections, sepsis, and peptic or carcinomatous ulceration constitute the primary disease. Streptococcus infection is most common. The colon bacillus may be present. Cases have been reported at every period of life between ten and ninety. Phlegmonous gastritis is four times as common in males as in females.

Symptoms.—In the circumscribed variety the symptoms are obscure. Pain may be absent. There may be a circumscribed tumor in the epigastrium, and vomiting of pus and blood. In the diffuse form the onset is sudden, with a rigor, severe prostration, and a rise of temperature to 104°–105° F. (40°–40.5° C.). Gastric symptoms speedily supervene. They consist of the urgent and continuous vomiting of pus, mucus, and bile, epigastric pain and tenderness, and, in rare instances, the signs of a fluctuating tumor. In the course of a brief period general abdominal tenderness, meteorism, and other signs of peritonitis usually appear.

The diagnosis has rarely been made *intra vitam*. The occurrence of the above symptoms in the course of a severe general infection or sepsis would be suggestive. The differential diagnosis concerns perigastritis following peptic ulcer, circumscribed peritonitis, acute pancreatitis, cholecystitis, and toxic gastritis. The prognosis is in the highest degree unfavorable. Of the reported cases 95 per cent. have terminated fatally.

3. **Diphtheritic Gastritis.**—Pseudomembranous inflammation of the gastric mucosa occurs as a true diphtheria of the stomach in cases of invasion by the Klebs-Löffler bacillus in diphtheria of the œsophagus, throat, or upper respiratory passages, but it is a very rare complication. More commonly it occurs as a complication of other infectious diseases, as enteric fever, pneumonia, the exanthemata, and sepsis. In children tuberculosis is often the primary infectious disease. A condition closely resembling diphtheritic gastritis may be produced by corrosive poisons. The characteristic lesion is the presence of the pseudomembrane, which may be diffuse, patchy, or arranged in irregular streaks extending from the cardia to the pylorus. The superficial layer is formed by a coagulation necrosis. The bacteriological findings vary. Streptococci, tubercle bacilli, and Klebs-Löffler bacilli have been more commonly isolated. The symptoms are not characteristic. Vomited membrane may have been dislodged from the upper air-passages, or the pharynx or œsophagus. The diagnosis, with a very few exceptions, has not been made during life.

4. **Parasitic Gastritis.**—Inflammation of the stomach as the result of infection by various pathogenic organisms by way of the blood and lymph stream—*infectious gastritis*—is of common occurrence. This form occurs in various septic conditions, enteric fever, pneumonia, and the exanthemata.

The presence of moulds and yeasts in lesions of the gastric mucosa renders it probable that, under certain circumstances, those agencies may cause or aggravate such conditions as inflammation, erosion, and ulceration. Among the growths obtained by lavage or observed post mortem are mucor mycelia, forms of leptothrix, thread fungi, Oidium lactis and albicans, favus, and Penicillium glaucum. The symptoms are not characteristic and their dependence upon the presence of fungi is uncertain. The subject is of anatomical rather than of clinical importance.

5. **Dietetic gastritis** is of common occurrence and great clinical importance.

Etiology.—Individual and family predisposition are common. Gouty subjects are peculiarly liable to subacute and acute gastritis.

The exciting causes comprise excesses at table, highly seasoned foods, unwholesome, indigestible, or tainted articles of food, large amounts of unduly cold or hot fluid, and alcohol.

Symptoms.—Habitual sensations of weight and gastric distress characterize the subacute forms. The more acute variety is associated with colicky pain, distention, fulness, and vomiting, which are often relieved by removing the offending material. In very acute cases the inflammation persists with the pain, colic, and hypersecretion of mucus and gastric fluid. Under such circumstances vomiting does not always afford relief. Bile frequently appears in the vomited matter. The early vomitus may contain undigested food, hydrochloric, lactic, and butyric acids, and have a butyric odor; the subsequent vomitings consist principally of water and mucus and are of a light green color. Diarrhœa may occur. Only in very severe cases is there fever. The tongue is coated, the pulse and general condition are usually good. Improvement commonly takes place in a few hours; occasionally an attack lasts two or three days. Slight fulness in the epigastrium and tenderness on pressure over the stomach are common in severe cases. Two or three days of persistent vomiting may be followed by retraction of the abdomen.

Diagnosis.—Direct.—Epigastric pain, nausea, and vomiting, following or referable to some indiscretion of the diet, justify a diagnosis of simple acute gastritis, particularly in the absence of fever or marked general symptoms. Fever, rapid pulse, and prostration, with persistent vomiting, should arouse the suspicion of an acute infection, cholecystitis, or intestinal or pyloric obstruction.

Differential.—If associated with fever at the onset, acute gastritis must be differentiated from, (a) *various infections,* as scarlet fever, meningitis. In acute gastritis the constitutional symptoms are less severe, there is absence of local or other phenomena peculiar to the specific infections, less intense pyrexia, and, as a rule, early improvement. (b) *Mild or Abortive Enteric Fever.*—Acute gastritis has a more rapid onset, an abrupt rise of temperature rather than the step-like rise, and rose spots, bronchitis, enlarged spleen, and diarrhœa do not occur. (c) Severe pain may suggest *gall-stone colic,* but the pain is usually less severe; the vomiting and pain of gastritis are more continuous; chilliness or a chill does not occur; and the general symptoms are less pronounced. Marked icterus is absent, though the possibility of an associated duodenitis with catarrhal jaundice

is to be borne in mind. (d) *Gastric Crises of Locomotor Ataxia.*—The Argyll-Robertson pupil, ataxia, and loss of knee-jerks are distinctive. If associated with persistent vomiting, acute gastritis must be differentiated from, (e) *pyloric obstruction* and *intestinal obstruction*. In both of these conditions the local signs are apt to be marked, serious general symptoms are present, and the history of the condition is different.

ii. Chronic Gastritis.

Chronic Gastric Catarrh.

Definition.—Chronic inflammation of the gastric mucosa, giving rise to mucus in excess and alterations in the gastric juice, associated with marked disturbance of the digestion and weakening of the muscular coat.

Etiology.—In many families there is a predisposition to chronic gastritis—chronic dyspepsia. Many constitutional diseases act as predisposing factors. Careless habits of eating and drinking, and the persistent use of gastric irritants are the chief causes. Chronic congestion of the stomach, the result of heart disease or hepatic cirrhosis, commonly ends in chronic gastritis, and most of the local gastric diseases, as cancer and ulcer, bring about the same condition.

Symptoms.—Headache, drowsiness, dizziness, inaptitude for work, and sallow complexion are common general symptoms. A coated tongue, bad taste in the mouth, aphthous stomatitis, chronic pharyngitis are usually present. A variable and capricious appetite, occasional repugnance for food, burning sensations in the œsophagus and at the cardiac end of the stomach—heart-burn—are early symptoms. Distress and weight in the stomach, oppression, distention, and actual pain (more particularly after meals), belching of gas, and eructations of bitter fluid soon occur if the condition persists. Nausea may be an early symptom. Vomiting usually appears late and occurs soon after eating or in the morning before food. That which occurs after a meal contains mucus in excess. Undigested food, indicating retention, fermentation of the carbohydrates, diminished amount of free HCl and ferments (or none at all), and traces of lactic and butyric acids are characteristic of delayed vomiting in rare cases. The vomitus has a sour odor. That occurring early in the day is composed of small amounts of thick mucus.

Diagnosis.—The direct diagnosis depends upon analysis of the gastric contents after a test-meal. There is an excess of thick, grayish mucus intimately mixed with the food residues and brought up with difficulty. The toast or bread may have passed out of the stomach completely, or it may remain in varying amounts mixed with mucus and poorly minced. Early in the disease free HCl may be normal in amount or even slightly increased; later it is diminished or absent. Lactic acid is not usually present. Both peptic digestion and the milk-curdling reaction for rennin may be absent. Further proof of muscular weakness and excess of mucus follows the washing of the stomach after the removal of the test-meal. The water used must often be removed by suction or siphonage instead of gushing back as is the case when poured into a normal stomach. It contains numerous mucous flakes, which accumulate in a stringy mass on the surface.

Inflation often shows dilatation at the lower border, reaching the level of the umbilicus.

Differential Diagnosis.— 1. *Ulcer of the Stomach.*—In chronic gastritis the pain is less intense and more continuous, less aggravated after food, and more diffuse. The decline in general health is less marked and rapid, and there is absence of hæmatemesis or blood in the stools. A coated tongue is common in chronic gastritis, while a clean red tongue is usually present in the hyperacid conditions in which ulcer occurs. There is no point of extreme local tenderness in chronic gastritis and no evidence of obstruction and muscular hypertrophy such as are sometimes demonstrable in chronic ulcer.

The results of gastric analysis in chronic gastritis and peptic ulcer are in strong contrast (see Gastric Ulcer). The chemical and physical conditions of the vomitus are, however, much modified when gastritis and ulcer are associated, as not rarely happens.

2. *Cancer of the Stomach.*—The differential diagnosis in the absence of tumor is at times most difficult. The loss of flesh and strength in chronic gastritis is rarely so rapid as in cancer. A protracted course is in favor of gastritis. Pain and vomiting are less marked, less persistent, and more amenable to treatment in chronic gastritis than in carcinoma ventriculi. For differences in the results of gastric analysis see Cancer of the Stomach.

3. *Pernicious Anæmia with Gastric Symptoms.*—The gastric condition is usually a chronic anacid gastritis. The differentiation rests upon the comparatively rapid and extreme deterioration of health and the blood examination.

4. *Gastric Neuroses.*—The conditions are frequently associated. The irregular dietetic habits and despondency characteristic of neurasthenia often cause chronic gastritis. Chronic gastritis is more amenable to dietetic treatment. Articles of food, such as tea, coffee, alcohol, and hot stimulating drinks, which aggravate the symptoms of gastritis, often allay the subjective symptoms of a neurosis. Regulation and restriction of the diet is usually beneficial in gastritis; not so, as a rule, in the neuroses. Fermentation and consequent flatulence and belching are more pronounced in gastritis; the flatulence of the neuroses is largely due to air swallowed or worked into the stomach. Anæmia is more marked in chronic gastritis than in the gastric neuroses.

A correct diagnosis may be made in most cases by the method of fractional gastric analysis and properly conducted serial röntgenological examinations. The anamnesis is important.

Prognosis.—Cases of chronic gastritis seen early and systematically treated get well. Advanced cases are comfortable only on a non-irritating diet and require continuous therapeutic management.

iii. Dilatation of the Stomach.

Gastrectasis.

Enlargement of the stomach, usually attended with weakening and thinning of the various coats, and supersecretion. Acute and chronic dilatations are recognized.

Acute Dilatation.—The **etiology** is not well understood. The condition is that of paralytic distention—angiomesenteric ileus. The stomach and duodenum are distended, with a patulous pylorus. It occurs as a post-operative complication in surgery of the stomach and colon and has been attributed to anæsthesia and general debilitating influences. Post-mortem findings point to acute dilatation as a terminal condition in acute illness, especially pneumonia and cardiac disease.

Symptoms.—Sudden collapse symptoms, the vomiting of enormous amounts of fluid, moderate pain, and pressure symptoms from distention, as dyspnœa and cardiac oppression, constitute the symptom-complex. The fluid reaccumulates as fast as vomited.

Physical Signs.—The physical signs of enormous distention of the stomach are present. The enlargement is often such that its true outline is lost. Peristalsis is not observed. The removal of large amounts of fluid, as much as eight or nine pints, by the stomach-tube and subsidence of the distention are, as a rule, followed by only transient relief of symptoms. This fluid is dark brown or clear and contains traces of blood and bile. Its odor is foul but not fecal. HCl is present in diminished but variable amounts, and there are in some cases traces of lactic acid. Constipation, oliguria, and torturing thirst not easily allayed are among the symptoms.

Diagnosis.—Direct.—Acute symptoms of collapse and oppression, epigastric pain, profuse vomiting of dark or clear fluid, marked distention of the stomach (the signs indicating fluid rather than gas), and the rapid reaccumulation of fluid after removal are the diagnostic criteria. The condition is not always recognized during life.

Differential.—*Acute Obstruction of the Pylorus and Upper Duodenum.*—Active peristalsis, moderate distention, and a relatively small accumulation of fluid, not immediately returning after withdrawal by vomiting or the tube, point to pyloric obstruction.

Acute Obstruction of the Duodenum below the Entrance of the Bile and Pancreatic Ducts or of the Intestine Still Lower Down.—The presence of considerable quantities of bile and pancreatic fluid in the vomitus or material removed would be significant. A fecal odor in the vomitus would suggest obstruction lower down.

Prognosis.—The condition is almost always fatal.

Chronic Dilatation.—Etiology.—There may be a family tendency. General and gastric debilitating conditions constitute predisposing influences. Habitual overdistention from excesses in eating or drinking may induce dilatation without pyloric obstruction being present. The majority of cases arise from obstruction at the pylorus, from cancer, ulcer with cicatricial contraction, adhesions to the gall-bladder, kinking of the duodenum, or stretching of the gastric walls already weakened by chronic gastritis.

Symptoms.—The condition may exist for an indefinite period without symptoms. In cases occurring independently of cancer or ulcer the loss of weight and strength is less rapid and anæmia and cachexia may not be present. Thirst, constipation, headache, torpor, and dizziness are common symptoms. Persistent dyspepsia, flatulency, belching, eructations, nausea, and eventually recurrent vomiting of large amounts of fermented undi-

gested food characterize the course of the affection. The dyspeptic symptoms resemble those of chronic gastritis, and comprise pain and weight immediately or shortly after eating, or hyperacidity four or five hours after the meal. Retention vomiting is common. Pain, in the absence of hyperacidity, is not usual except in carcinoma or ulcer.

Physical Signs.—Thinning of the abdominal wall is usually present with the general wasting. The skin is often dry and harsh. *Inspection.*—The abdomen may show general fulness, or the epigastrium, hypochondria, or supra-umbilical region may be especially prominent. The stomach may be distinctly outlined, its greater curvature sometimes reaching into the pelvis. Displacement of the pylorus and lesser curvature may outline the whole stomach as a dilated sac lying in great part below the umbilicus. Peristalsis from left to right is easily distinguished. In obstructive cases it ends in the hypertrophied pylorus or tumor mass. On palpation "clapotage" or splashing of fluid is readily obtained when the dilatation reaches the umbilicus. The cushiony condition of the air-distended stomach is readily felt. Percussion yields tympany. In the erect posture the lower limit of the stomach can be determined by the dulness of the contained fluid. Auscultation reveals nothing important.

Inflation with air through the tube or by CO_2 shows distention reaching to the umbilicus or below it. The whole stomach can often be outlined. Usually, however, the outline obtained does not include the lesser curvature. *Auscultation.*—The cask-like tympany produced in the distended stomach by quickly compressing or relaxing the bulb of the tube, or by percussing with the finger, is readily heard with the stethoscope and can be followed over the whole stomach, being lost on passing the stethoscope's bell away from the immediate area of the stomach. Inflation with water has no practical value.

Large barium meals are necessary in the study of the outline of the dilated stomach by röntgenological methods. Both recumbent and erect positions must be employed. Transillumination can at best show only the lower border of the stomach.

The vomitus is variable. That of dilatation consequent upon chronic gastritis shows mucus, usually absence of free HCl, marked fermentation; that associated with cancer frequently has an odor of putrefaction in addition to that of fermentation; the vomitus in which much free HCl is present is rarely foul, the HCl acting as an antiseptic. The amount may be enormous.

The test-meal removed in an hour may come away with several hundred c.c. additional fluid. Much mucus is present in chronic gastritis. The toast is poorly minced and almost wholly returned. Free HCl may be present in excess, even in very advanced cases, or may be absent, as in cases of cancer or gastritis. Lactic acid may be persistently present in cancer. It tends, however, to disappear under systematic lavage. The weakness of the gastric muscle is shown by the necessity of using suction and siphonage to remove the contents of the stomach.

Diagnosis.—DIRECT.—Persistent dyspepsia, flatulence, eructations, and vomiting of large amounts of long-retained fermented food remains are suggestive. Distention, the outlining of an enlarged displaced stomach

visible peristalsis, and an abnormally large amount of gastric contents after a test-meal are conclusive. Inspection alone may make the diagnosis. A pyloric tumor with its associated signs suggests gastrectasis.

Differential.—The anamnesis distinguishes dilatation of the stomach from cystic conditions of the mesentery, gall-bladder, and ovary, and from chronic dilatations of the colon. The last is not associated with "retention vomiting"; it shows the intestinal outlines and peristalsis from right to left, and can be reduced by passage of the rectal tube. The use of the stomach-tube is of great diagnostic importance.

Prognosis.—Dilatation of the stomach, when recent and unattended by obstruction at the pylorus and not excessive, may permanently subside. Many cases require systematic lavage. Operative measures are required for the relief of marked dilatation with pyloric obstruction.

iv. Gastric Ulcer.

Ulcus Ventriculi; Peptic Ulcer.

Definition.— Ulceration of the gastric mucosa in any part of its extent, due to nutritional disturbance in a circumscribed region and the action of the gastric juice. The necrotic areas may occur in the lower end of the œsophagus and in the duodenum as low as the papilla of Vater. They involve the various coats of the stomach and sometimes perforate. They are usually round or oval, with clean-cut edges in the acute and irregular indurated borders in the chronic cases. As a rule there is only one ulcer, but two or more are occasionally seen. The usual site is the posterior wall or the lesser curvature. Healing ulcers result in cicatricial deformities as pyloric stenosis or hour-glass contraction. A cancerous growth is by no means infrequent—*ulcus carcinomatosum*. Perforation may be followed by general peritonitis or subphrenic abscess, but the occurrence of adhesions when the ulceration is deep often prevents these accidents.

Etiology.— Anæmia and chlorosis predispose to the affection. The disease is more common in the working classes, in women than in men, and between twenty and forty than at other periods of life. Heredity has some influence. Hyperacidity of the gastric juice is usually present. Trauma or large superficial burns may be direct causes. Septic cases occur.

Symptoms.—General symptoms are often absent. Loss of weight and strength, and progressive anæmia, often of extreme degree, are common. Constipation is usual. Simple dyspepsia, distress and fulness after eating, flatulence, and belching are common symptoms. In such cases there is often normal acidity. In the more severe cases the dyspepsia is more intense; nausea and vomiting occurring three or four hours after eating, or severe agonizing pain on taking food and lasting two or three hours or more, or not beginning until the height of gastric acidity is reached, two or three hours after the meal, are frequent symptoms. At times the pain is gnawing in character, more marked when the stomach is empty, and relieved by food. Cases of this type usually show a high degree of acidity.

The pain is referred to the epigastrium, often radiating to the back and sides. Vomiting may give relief. Hemorrhage is common and may be the first symptom. The blood may be passed either by the mouth or bowel. Concealed hemorrhage may occur with characteristic symptoms. Recurrent hemorrhage may end fatally. I have seen a single profuse hemorrhage followed by death in a man in apparent health. Small continued hemorrhages may only be recognized by blood tests. The testing for occult blood in the stools is most important in such cases.

The symptoms may continue for years. Perforation of the stomach and peritonitis may be the first clear indications of an ulcer. The local symptoms in old cases with pyloric obstruction are those of gastrectasis, flatulence, dyspepsia, nausea, and retention vomiting. In early cases, in the absence of obstruction there may be nothing to indicate disease of the stomach. In others tenderness in the epigastrium, narrowly circumscribed, may be the only symptom. During the inactive stage of an ulcer near the pylorus or in the duodenum temporary signs of pyloric obstruction may develop. These are probably due to spasmodic closure of the pylorus and the infiltration of the neighboring tissue. Fulness of the epigastrium, the outline of the enlarged stomach, and visible peristalsis may be evident. The thickened and hypertrophied pylorus can sometimes be felt during contraction. Tenderness over the pylorus is often marked. In chronic cases the physical signs indicate tumor formation due to scarring or puckering of the ulcerated area, thickening and muscular hypertrophy, and hourglass contraction. These are often much more manifest upon inflation.

Gastric Analysis.—The vomitus or gastric contents from the fasting stomach, usually thin, watery, and light green in color, frequently contain large amounts of free HCl. The gastric contents removed after the ordinary test-meals are usually in excess of the normal and generally contain some mucus and a small amount of well-mixed food residue. They show a high total acidity, a high degree of free HCl, pepsin and rennin, no lactic or butyric acid. Traces of blood by chemical tests are common. Under the microscope small round cells and pus-cells can be demonstrated in most cases when the ulceration is active. The red blood-cells are, as a rule, disintegrated by the high acidity. In the older cases, the same high percentage of free HCl and hypersecretion may sometimes be found in the fasting stomach—gastrosuccorrhœa or Reichmann's disease. The vomitus in cases with obstruction is sometimes of enormous quantity, containing free HCl and mucus. It is acid in odor but rarely foul. The high total acidity and free HCl are occasionally absent in well-marked cases of gastric ulcer.

Diagnosis.—Direct.—The diagnosis rests upon persistent dyspepsia, anæmia with loss of weight and strength, marked circumscribed tenderness in the epigastrium, a high degree of gastric acidity, and occult blood in the fæces, gastric contents, or vomitus. In marked cases intense pain after eating, vomiting, nausea, the sudden appearance of hæmatemesis or tarry stools, with rapidly developing weakness and anæmia occur. In long-standing cases the symptoms and signs of pyloric obstruction or gastric malformation are significant. For X-ray findings see Vol. I.

Differential.—1. *Chronic Gastritis.*—In ulcer the high degree of acidity of the gastric juice, the presence of occult blood in the gastric contents and fæces, the more marked tenderness or local pain are distinctive. A well-defined hæmatemesis or the passage of blood per rectum is conclusive. 2. *Gastric Superacidity.*—Loss of weight is common to both; anæmia is more common in ulcer. Localized pain is more marked in ulcer; nausea and vomiting, pain after eating, hæmatemesis, tarry stools, occult blood in the fæces or gastric contents are not symptoms of superacidity. Relief of pain on taking food is suggestive of simple hyperacidity, in which signs of obstruction and marked retention do not, as a rule, occur. 3. *Carcinoma of the Stomach.*—Sudden onset with hæmatemesis, hemorrhage from the bowels, and perforation are suggestive of ulcer. The history of many years' duration is against cancer. Cachexia develops rapidly in cancer, is unusual in ulcer. Hæmatemesis or hemorrhage from the bowels is common in ulcer, as in hyperacidity of the gastric juice, while the presence of lactic and butyric acids is unusual. Absent or diminished free HCl is common in cancer, the presence of lactic and butyric acids frequent.

The signs of a tumor are, as a rule, present early in cancer, but late if at all, and associated with scar formation, in ulcer. Pyloric obstruction may be an early condition in cancer.

Duodenal ulcer is more commonly attended by right-sided pain and tenderness, rigidity of the right rectus muscle, periodicity of symptoms and there is a difference in the X-ray findings. Cholecystitis and biliary calculus bear only a remote resemblance to *ulcus ventriculi*, and are attended by characteristic colic, occasionally urinary bile pigments, and jaundice and the absence of hæmatemesis and tarry stools or occult blood in the fæces. In an obscure case an infected high appendix, renal calculus, intestinal parasites, epigastric hernia and gastric crises are to be considered.

v. Cancer of the Stomach.

Carcinoma Ventriculi.

Definition.— Cancerous infiltration of the stomach walls occurring most frequently toward the pylorus and about the lesser curvature.

Etiology.— The predisposition has been thought to be hereditary. A chronic gastric ulcer not infrequently becomes the seat of a carcinoma. Middle and advanced life is the time of common occurrence, but there is no "age of cancer." Irritation of the stomach by improper diet, injury, or pressure from without have all been considered exciting causes.

Symptoms.— Rapid anæmia, loss of strength and weight, and early developing cachexia in a middle-aged person are suggestive. Persistent distress in the stomach after eating or even when the stomach is empty, often amounting to intense pain, is an early symptom. Nausea and anorexia soon follow. Vomiting may be an early symptom. If there is marked obstruction at the pylorus with consequent dilatation, the vomitus may be of large volume and foul odor. The vomiting of altered blood ("coffee-

ground vomit'') and passage of small amounts of blood in the stools occur. Constipation is often present.

Physical Signs. — Inspection may show nothing, especially in early cases. Metastasis may be seen in the supraclavicular fossa or beneath the skin of the abdomen. Fulness of the epigastrium and left hypochondrium, visible outline of the stomach, displaced and distended visible peristalsis (usually from left to right), and distinct contracting tumor may be observed. Inflation will often produce peristalsis and bring a tumor mass, not otherwise recognizable, into evidence. Inflation will often show the abnormal size and position of the stomach. Palpation also may reveal nothing at first, especially if the tumor is posterior or adherent. Careful and repeated palpation, aided by ''dipping'' and slapping the abdomen, may reveal the presence of a new growth. Inflation aids palpation at times by bringing the tumor into the field of examination. The movements of the tumor, with respiration, with movements of the body, with the aortic pulsation, and from the muscular contraction, should be noted.

Gastric Analysis.—Vomitus or a test-meal containing traces of blood and showing no free hydrochloric acid and a lowered total acidity must be regarded as suspicious in all cases. Lactic acid is usually to be found in the vomitus after a mixed meal if there is any degree of stagnation, and in the test-meals given in late cases. After a few days' lavage it diminishes and may disappear. Butyric acid will be found in the vomitus under the same conditions as lactic acid. Pepsin and rennin are usually to be found. Pieces of cancer tissue should be looked for. Oppler-Boas bacilli are found more frequently in the vomitus of cancer than in any other condition. They disappear quickly under lavage. Pus-cells, sarcinæ, yeast cells, and many bacteria may be seen. Free HCl is not always absent, and may even be present in excess. With obstruction at the pylorus the amount of material obtained after a test-meal will be excessive.

Diagnosis.—Direct.—Persistent dyspepsia, pain, loss of weight and strength, anæmia, a tumor in the gastric area, signs of displacement and dilatation of the stomach, absence of free HCl, presence of lactic acid, and Oppler-Boas bacilli in the test-meal or vomitus, and excessive amount of vomitus, and gastric contents with evidences of stagnation are the main points in the diagnosis. X-ray examinations in suspected cases are imperative.

Differential.—*Chronic Gastritis* (see Chronic Gastritis). *Pernicious Anæmia.*—Gastric symptoms with absence of free HCl often occur, but this condition may be distinguished by the blood examination. The anæmia of cancer rarely falls as low as in pernicious anæmia. The color index is low in cancer. There may be a slight leucocytosis, and the presence of megaloblasts is rarely noted. In general the blood picture is that of a secondary anæmia. Hæmatemesis, tarry stools, the reaction for occult blood in the fæces and gastric contents are indicative of a gastric lesion. *Severe Secondary Anæmia with Gastric Symptoms.*—The presence of lactic acid in the test-meal, the absence of free HCl, the presence of a tumor, and occult or gross blood in the gastric contents and fæces point to cancer.

Prognosis.—Unless an early diagnosis makes a total extirpation of

the tumor possible the course is steadily downward. Gastro-enterostomy may be of service. Temporary improvement usually follows the institution of lavage in cases that have marked obstruction at the pylorus.

vi. Hypertrophic Stenosis of the Pylorus.

Definition. — Obstruction of the pylorus from hypertrophy of the musculature and the submucous tissue.

Etiology. — Most of the reported cases are in infants and are congenital; even those reported in adults are considered to be late developing cases. The same conditions that induce pyloric spasm, namely, nervous and direct irritation, are possible exciting causes.

Symptoms. — The symptoms and physical signs are those of obstruction at the pylorus.

Diagnosis. — DIRECT.—Symptoms of obstruction at the pylorus, visible peristalsis, and palpable pyloric tumor occurring in infants are diagnostic of the condition. In adults the condition can hardly be distinguished from simple pyloric spasm.

DIFFERENTIAL.—There is no other condition which can be confused with hypertrophic stenosis in infants except pyloric spasm. Relief following general and dietetic treatment would justify the diagnosis of the latter affection. True hypertrophic stenosis in adults cannot always be distinguished from other forms of pyloric tumor or obstruction. It may be differentiated from spasm of the pylorus by the fact that the latter is not followed by permanent lesions, as tumor or dilatation. Its occurrence in early infancy is against its being confounded with either cancer or ulcer. In adult cases the absence of anæmia and wasting, cachexia, hemorrhage, and occult blood are of diagnostic value.

vii. Tuberculosis of the Stomach.

Tuberculous ulceration of the stomach is discussed under Tuberculosis.

viii. The Gastric Neuroses.

The gastric neuroses, nervous disorders of the stomach, "nervous dyspepsia," include a host of conditions many of which are explained and described by their names. With a few exceptions, which comprise the nervous disorders of secretion, they are characterized by the predominance of symptoms over physical signs. Disorders of sensation, secretion, and of the motor function are recognized.

1. **Disorders of Sensation.** — (a) **Gastric Hyperæsthesia.** —A condition of oversensitiveness of the gastric mucous membrane. ETIOLOGY.—The general nervous state is the predisposing influence. Any article of food may be the exciting cause. Overacidity of the gastric juice in an empty stomach is a frequent cause. SYMPTOMS.—Neurasthenia or hysterical symptoms are common; wasting or anæmia is infrequent. Gastric distress, gnawing sensations, burning in the stomach immediately or shortly after taking food or when the stomach is empty are the main features. The sensation seems to be that of feeling the stomach doing its work.

Stimulants or condiments often relieve the symptoms. There is often tenderness over the stomach. The gastric contents after a test-meal usually yield normal results. Diagnosis.—*Direct.*—Hysterical or neurasthenic manifestations, gastric symptoms occurring before there has been time for acid to accumulate or for fermentation to begin, negative physical examination and negative results of the gastric analysis are suggestive. *Differential.—Gastric Hyperacidity.*—The gastric analysis sometimes shows slight hyperacidity; as a rule, the analysis serves to distinguish simple hyperæsthesia from hyperacidity. The symptoms in hyperacidity develop later—three to four hours after a meal. *Chronic Gastritis.*—The sensory symptoms in some forms of chronic gastritis are practically the same but they are definitely related to certain articles of diet. The gastric analysis of chronic gastritis shows distinct differences (see Chronic Gastritis). *Gastric Ulcer.*—Hyperæsthesia rarely produces anæmia or wasting. There is no vomiting, no hæmatemesis or tarry stools, no signs of pyloric obstruction, no occult blood in fæces or gastric contents, and usually no hyperacidity of the gastric juice. *Gastric Cancer.*—Absence of anæmia and cachexia, absence of vomiting, hemorrhages, gross and occult, absence of a tumor or signs of dilatation and obstruction, a normal condition of the gastric juice favor the diagnosis of hyperæsthesia.

Prognosis.—Recovery depends upon the course of the general nervous condition. Persistence of the symptoms may reduce the patient to an extreme degree.

Anorexia nervosa is considered in the sections on hysteria and neurasthenia. Excessive hunger, bulimia, absence of the sense of repletion—acoria—are often merely symptoms of hyperacidity. They may occur, however, with a normal condition of the gastric juice.

(b) **Gastralgia; Gastrodynia.** — Severe paroxysmal pain in the stomach, often periodic, not referable to ulcer, cancer, or organic nervous disease (gastric crises of tabes). This is often symptomatic of excessive acid secretion. Etiology.—The predisposing influence is always neurotic. An exciting cause cannot always be found, the gastralgic attacks occurring more or less independently of food ingestion. Symptoms.—There are almost always general nervous symptoms. Acute grinding pain in the epigastrium and gastric region, which radiates to the back, is the main feature. Vomiting is unusual. Pressure may or may not relieve the pain. Tenderness on deep pressure is the rule. Diagnosis.—Acute periodic painful attacks with no evidence of gastritis, ulcer, hyperacidity, or of locomotor ataxia. Marked general nervous symptoms are suggestive. The *differential diagnosis* concerns cancer, peptic ulcer, the gastric crisis of tabes, and biliary colic. *Cancer and Ulcer.*—The nervous symptoms and the general condition of the patient in gastralgia are important diagnostic points, since many cases of gastralgia show absence or even excess of HCl in the gastric juice. Negative occult blood-tests of the fæces and gastric material, absence of anæmia and cachexia, of tumor mass and physical signs are in favor of gastralgia. *Gastric Crisis of Tabes.*—Absence of the knee-jerk and other tabetic phenomena are conclusive. *Gall-stone Colic.*—The distinction is sometimes extremely difficult, since the pain of gall-stone colic may be

referred to the epigastrium, subsequent jaundice may not occur, and the calculus may be masked in the faeces or not passed from the duct. Prognosis.—The prognosis in gastralgia is usually favorable. It depends upon the course of the general nervous condition.

2. **Disorders of Secretion.**— Three main varieties are recognized: supersecretion, hyperacidity of the gastric juice, and hypoacidity and anacidity.

Either hyper- or hypoacidity may be associated with supersecretion; hyperacidity and supersecretion is the more frequent combination.

(a) **Supersecretion; Gastrosuccorrhœa.**—A condition in which excess of gastric juice is secreted continuously,—*Reichmann's disease*,—or periodically,—*Rossbach's disease; nervous gastroxynsis*,—usually in association with a certain degree of dilatation of the stomach from a general relaxation of its muscular tone. Etiology.—General neurasthenia is the main predisposing cause. Stimulants and tobacco are occasionally exciting causes. Symptoms.—Neurasthenic symptoms are almost always present. In the periodic form there occurs suddenly the accumulation of large amounts of fluid, associated usually with gnawing distress or even pain, eructations or vomiting of a clear watery fluid ensue and may persist for several days. The secretion is enormous and independent of food, the symptoms often occurring in the early morning. If hyperacidity is present the irritation of œsophagus and pharynx may occasion great distress. The continuous form ends frequently in dilatation of the stomach from the persistent pyloric spasm so often present and the weight of the accumulating fluid upon the relaxed muscles. Physical Signs.—Dipping palpation may give clapotage. Percussion in the erect position may show a level of fluid in a position lower than normal. The stomach-tube, passed in the morning or at other times when the stomach is presumably empty, may bring away as much as 200 to 300 c.c. of fluid, often highly acid from free HCl. Inflation shows a moderate degree of dilatation. Spasm of the pylorus and peristalsis may be seen and felt. The test-meal may be returned after an hour with an accumulation of several hundred c.c. of a high or normal degree of total acidity and with a large or normal amount of free HCl. Mucus is not in excess; lactic acid is not present. In late cases of continuous supersecretion the condition is practically that of dilatation. Diagnosis.—*Direct.*—The continuous or periodical presence in the stomach of an excess of fluid having the above characters and its accumulation independently of the stimulation of food are the main characteristics of the condition. *Differential.—Acute Gastritis.*—Acute gastritis usually has a distinct and recognizable etiology. The fluid vomited is smaller in quantity, usually anacid, and contains mucus. *Acute Dilatation.*—In acute dilatation the general symptoms are marked from the outset. The condition is much more serious, and neither vomiting nor lavage has much effect upon the course of the attack. *Chronic Dilatation.*—Supersecretion often ends in dilatation. Simple supersecretion in its early stages, however, has a different history. Retention vomiting and the physical signs of marked dilatation do not occur. Prognosis.—This depends upon the nervous state; as a rule it is good.

(b) **Hyperacidity; Hyperchlorhydria.** —Excess of free HCl in the gastric juice. ETIOLOGY.—Hysteria and general neurasthenia predispose to the condition. Irregular habits of eating, stimulants, tobacco, and an excess of proteid nourishment are common exciting causes. SYMPTOMS.—The general nervous symptoms of the underlying condition are present. Headache, hunger, and constipation are common. Gastric hyperæsthesia is frequently present. Supersecretion often coexists. Gnawing distress, burning, or severe pain developing two to four hours after eating, relieved in turn by eating proteid food, are local symptoms. When vomiting occurs the symptoms are usually relieved. The tongue is commonly clean, red, and moist, and there is epigastric tenderness. The test-meal is usually expelled vigorously and in excessive quantity, as some degree of supersecretion is almost always present. The digestion of the starch is retarded. The total acidity, instead of a normal 40, may reach 120 or 150. Free HCl may be as high as 90 to 110. If the condition has been persistent mucus may be present. Lactic acid is absent. Many round epithelial cells showing mitosis may at times be seen under the microscope. The vomitus on account of its high HCl percentage does not readily ferment. Bacteria are not present in any large numbers. Meat digestion can be shown to be rapid. DIAGNOSIS.—*Direct.*—Distress two to four hours after eating, relieved by taking food, a high degree of free acid in the vomitus, and the results of the chemical examination of the gastric contents constitute the basis of a direct diagnosis. *Differential.—Ulcer.*—Hyperacidity of the gastric juice is common in ulcer. Pain immediately after eating is usual in ulcer, and vomiting is more common. Local tenderness or pain, a history of hæmatemesis or tarry stool, loss of weight, anæmia, occult blood in gastric contents and fæces are important. The two conditions often coexist, the ulcer being unrecognized. Laboratory methods are imperatively required in doubtful cases. *Cancer,* with normally acid or hyperacid gastric juice. The general symptoms, as loss of weight, cachexia, anæmia, the persistence of local symptoms, as vomiting and pain, are distinctive of cancer, while occult blood in the fæces or gastric contents, or gross hemorrhages are against the diagnosis of simple hyperacidity. The diagnosis may, in rare cases, be impossible until a palpable tumor is detected or a dense shadow appears in the skiagram. PROGNOSIS.—The condition may last for years. Relief under appropriate treatment is the rule.

(c) **Hypoacidity; Anacidity; Hypochlorhydria.** —Conditions of the gastric juice in which the free HCl is of low value, or lacking. ETIOLOGY.—Hysteria and depressed nervous states are predisposing causes or even the exciting cause. It is to be remembered that the Ewald test-meal may fail to provoke much secretion. Low HCl in organic disease occurs as follows: in subacute and chronic gastritis, early carcinoma, dilatation of the stomach, and various chronic diseases of the abdominal viscera. SYMPTOMS.—Symptoms are frequently absent. Fermentation, flatulence, a feeling of fulness, or other manifestations of indigestion occur. Diarrhœa, anæmia, and pallor are more common than in the other secretory disorders. Local signs are absent. A coated tongue is usual. Inflation reveals nothing. The test-meal is brought away either as normal amount, or thick and pasty

from lack of fluid. Starch digestion has progressed. There is persistently a low degree of free HCl, and the total acidity is also low, or there may be no reaction for either acidity or free acid. Lactic acid is absent. Mucus is not in excess. Pepsin and rennin are present. When the gastric secretion is completely absent, as occurs in rare instances, the condition is designated "achylia gastrica." Fractional analysis is necessary.

Diagnosis.—*Direct.*—Persistent absence of free HCl, or its presence in an abnormally low percentage half an hour or one and a half hours after the test-meal, is suggestive. General nervous symptoms and the absence of localizing gastric phenomena are important. *Differential.—Chronic Gastritis.*—The relation of symptoms to food is more marked in gastritis. Dyspeptic symptoms are more marked, vomiting and nausea more pronounced in gastritis, and mucus in excess is common. Nervous symptoms are not especially prominent in gastritis. *Cancer.*—In cancer constitutional symptoms are more marked. Pain, vomiting, nausea, occult and gross hemorrhages, anæmia, wasting, physical signs of tumor and dilatation may be present. The test-meal may show no distinguishing feature. The presence of lactic acid and blood is in favor of cancer. *The Anacidity Seen in Pernicious Anæmia.*—The blood picture is the only means of differentiation; the gastric conditions are undistinguishable.

Prognosis.—The condition often persists unrelieved. Occasionally the secretion of free HCl may be restored.

3. **Disorders of the Motor Functions.**— With one or two exceptions disorders of the motor functions of the stomach are the expression of hysteria or are cultivated habits. They are usually not associated with gastric distress and show no physical signs or changes in the gastric juice. Some of them, as rumination, peristaltic unrest, gurgling, and singultus, need no special description here. Nervous vomiting, relaxation of the cardiac orifice, is unattended by nausea or symptoms of irritation. The ease with which the gastric contents are regurgitated and the absence of the signs of fermentation, hyperacidity, etc., are of diagnostic importance.

Spasm of the cardiac orifice may be differentiated from œsophageal stricture by the use of œsophageal sounds and bougies. The regurgitation of unaltered food, without the chemical reaction of gastric juice, is characteristic of stricture or diverticula of the œsophagus.

Spasm of the pylorus is frequently associated with hyperacidity. Gastric distention, flatulence, belching, colicky pain, are common associated symptoms. In thin subjects the contracted pylorus can be seen and felt. Visible peristalsis may occur. The diagnosis from organic disease may be difficult. The good general condition of the patient, the absence of severe local signs, a normal or excessive acidity of the gastric juice are in favor of a neurosis.

Relaxation of the pylorus occasionally occurs, the stomach emptying itself almost at once. The condition is usually discovered acidentally during attempts at removing a test-meal.

Gastric Atony.— Atony of the gastric muscles is usually part of general muscular relaxation seen in nervous and exhausted states. It is practically a condition of moderate dilatation without pyloric obstruction. The

coexistence of neurotic symptoms or other disease is important in its recognition.

The prognosis of the motor neuroses of the stomach depends upon the underlying nervous condition and is usually good.

ix. Gastroptosis.

Definition.— A prolapse of the stomach from its natural position, due to stretching of its ligamentous attachments, usually associated with ptosis of other organs, especially the kidneys, liver, and large intestine.

Etiology.— The predisposing influences are neurasthenic states with weakened and relaxed musculature. Possibly the bad carriage and slouching forward of many neurasthenics who always require "straightening up" may be an exciting cause. Repeated pregnancies, recurring ascites, stretching, relaxing, and diastasis of the abdominal muscles are frequent causes.

Symptoms.— Cases in which the neurotic element is absent may show no symptoms, even with the stomach far out of place. As the stomach drags downward, however, kinking at the duodenum is likely to occur, since duodenal ptosis does not develop to any marked degree, and symptoms of mild obstruction may develop. In the markedly neurotic, gastroptosis once established seems to give rise to or keep in continuance many general symptoms, such as faintness, weakness, continued exhaustion, headache, depression, dragging pain in the back and abdomen, loss of weight and strength, sallowness, and slight anæmia. Flatulence from slight obstructive kinking at the duodenum, various forms of nervous dyspepsia, constipation, colicky abdominal pain are common symptoms. Persistence of the ptosis and obstruction, eventually causing chronic gastritis and distinct dilatation as well, will give rise to the symptoms characteristic of these conditions. Symptoms due to ptosis of the liver, kidneys, and transverse colon are usually coexistent.

Physical Signs.— Marked relaxation and thinning of the abdominal walls may be evident. If the patient lifts his head and shoulders from the couch, "diastasis recti" can often be easily made out. Peristalsis of the stomach or intestine is readily seen between the two flat band-like recti muscles. If much flatulence is present the stomach can be seen outlined and occupying the umbilical region or even reaching into the pelvis, the lesser curvature falling as low as the umbilicus. Palpation shows whether or not other organs, as the liver, kidneys, or spleen, are displayed, and on "dipping" whether or not clapotage is present. Palpable peristalsis is not as evident as in true obstructions, though the pylorus in contraction can frequently be made out. The relaxation and thinning of the abdominal walls is also readily appreciated by the touch. Auscultatory percussion yields gastric tympany practically normal in outline but completely displaced downward. Inflation gives characteristic results. The stomach is entirely displaced downward; the cardiac portion stretched downward; the lesser curvature clearly outlined about the umbilicus in more or less natural relationship to the greater. The stomach may show but little or no evidence

of dilatation. The test-meal may show normal acidity of the gastric juice and normal amount of free HCl or hyperacid, hypoacid, or anacid juice. There is usually some degree of retention and evidence of poor action of the gastric muscles, the bread or material used not being well minced or digested. Mucus may or not be present. The passage of food from the stomach, and absorption are delayed, as can be demonstrated by the potassium iodide test.

The X-ray examination is an important aid in the diagnosis of gastroptosis.

Diagnosis.—DIRECT.—Gastric and neurasthenic symptoms, displacement of liver and kidneys, relaxed abdominal walls, presence of the stomach in an abnormally low area, the organ remaining more or less normal in size and shape, the lesser curvature in the usual relation to the greater, are characteristic of the condition.

DIFFERENTIAL.—*Dilatation of the Stomach.*—Gastrectasis and gastroptosis often coexist. Symptoms such as nausea, copious vomiting, thirst, and wasting are significant of dilatation. So also are visible and palpable peristalsis and the signs of hypertrophied musculature. In dilatation the enlargement principally displaces the greater curvature downward, the lesser curvature remaining more or less fixed except in the case of movable tumor involving the pyloric extremity. The gastric contents removed in cases of dilatation are usually excessive, 300–600 c.c., whereas in gastroptosis there may be but a few c.c. more than normal.

Prognosis.—A markedly prolapsed stomach probably never returns to its former position. Untreated cases usually terminate in dilatation or become complicated with chronic gastritis. In many cases a surgical procedure is necessary to maintain the stomach in a position to properly empty itself.

II. DISEASES OF THE INTESTINES.

i. Enteritis.

Inflammation of the intestines. Any part or the whole of the gut may be involved. There are important clinical and pathological distinctions between inflammation of the large and of the small intestine.

Catarrhal Enteritis.—Definition.— A disordered condition of the small intestine associated with increased secretion and frequent watery or softened stools. Abdominal pain, mucous stools, and evidences of disordered intestinal digestion occur. Acute and chronic forms are described.

Etiology.— PREDISPOSING INFLUENCES.—Certain individuals are especially liable to catarrh of the bowels as a result of either dietetic or climatic conditions. In women and children intestinal catarrh occurs very readily. Habitual dietetic errors and chronic disease predispose to the development of catarrhal enteritis. EXCITING CAUSE.—Frequent attacks of acute enteritis may lead to a chronic catarrhal condition. Unwholesome food, toxic food products, certain poisons, as arsenic and mercury, nervous irritability, gastric disorders, particularly hyperacidity, intestinal affections,

tubercular ulceration, enteric fever, excessive use of purgatives, and sudden changes from a warm to a cold temperature are exciting causes. Variations in the composition of the intestinal juices, arising independently or due to lack of proper stimulus on the part of the gastric juice, may play an important part.

Symptoms.—General symptoms, such as depression, exhaustion, thirst, anorexia, nausea, are common to acute and chronic enteritis. Wasting occurs very rapidly in the former, and may be accompanied by fever.

Abdominal colic is more common in acute than in chronic enteritis. It is apt to occur shortly after eating and is usually referred to the mid-abdomen. Abdominal tenderness is commonly present. In the chronic cases pain is not a conspicuous symptom.

Diarrhœa is the main feature of the disorder. It may exist as, (a) frequent, watery, brownish colored, unoffensive acid movements, well mixed with brownish sago-like soft mucus. The absence of fetor is largely due to the fact that time for fermentation and decomposition is lacking. Microscopical examination shows excess of undigested food remnants. On standing such stools show gas formation and develop an intense putrefactive odor. This particular form of diarrhœa usually attends the acute cases. (b) Less frequent soft mushy stools, often distinctly pale and putty-like (pultaceous stools), very offensive when passed. At times no undue frequency is to be observed. There is an intimate mixture of fine sago-like mucus. The pallor is due to undigested fat; the mucus and fine gas bubbles to fermented carbohydrates. Microscopically undigested food particles are abundant. Fat droplets, fatty acid crystals, soaps, starch, meat fibres are in excess. Bile-stained epithelium and mucus can be seen. The reaction is intensely acid. Gas production occurs on standing. Neither occult nor gross blood is to be detected in uncomplicated catarrhs.

Fetor of the breath and a pasty tongue are common. In thin subjects visible peristalsis may be seen. The pulse is usually slow. The urine is diminished in amount and contains indican, very frequently also albumin and casts.

Diagnosis.—Abdominal pain and tenderness, diarrhœa, the presence in the movements of fine particles of bile-stained mucus and excess of undigested food are significant. Fermentation, acidity and pallor of the stool are indicative of intestinal catarrh. Occult or gross blood would indicate ulcerative or hemorrhagic conditions. Large flakes or masses of mucus occur in membranous enteritis. Time and the Widal reaction will distinguish the cases with fever from typhus abdominalis.

Prognosis.—In the acute cases the outlook is mostly favorable. Repeated attacks of acute enteritis may end in the chronic form. Even protracted cases occasionally terminate in recovery.

Phlegmonous Enteritis.—Acute suppurative inflammation of the submucous tissue of the small intestine, occurring anywhere in its course, either as a primary disease or as a secondary affection in various intestinal accidents.

I. **Primary Phlegmonous Enteritis.**—A disease of very rare occurrence. Etiology.—*Predisposing Influences.*—We know nothing of the conditions likely to induce primary phlegmonous enteritis. *The Exciting Cause.*—Acute bacterial infection by pyogenic organisms, usually the Streptococcus pyogenes or the Bacillus coli communis. Symptoms.—The symptoms are those of acute peritonitis. There is no definite classical picture of the disease. Diarrhœa is not a necessary accompaniment. Diagnosis.—The diagnosis has not been made during life. Prognosis.—The disease is invariably fatal.

II. **Secondary Phlegmonous Enteritis.**—A condition occurring in connection with various intestinal disorders. Embolism, carcinomatous and tuberculous ulceration, intussusception, strangulation may be complicated by phlegmonous enteritis. Etiology.—*Predisposing Influences.*—The above conditions predispose. *Exciting Cause.*—Infection by pyogenic organisms as the result of an infected embolus, thrombosis, extensive ulceration, or complete obstruction of the intestine from any cause. Symptoms.—The symptoms are those of the primary disorder plus those of peritonitis. There are no distinguishing features of the disease. Diagnosis.—The symptoms of peritonitis superadded to those of the existing intestinal condition may suggest phlegmon formation. The differentiation between developing phlegmonous enteritis and peritonitis is not possible. The prognosis is lethal. Recovery from phlegmonous inflammation does not occur.

Diphtheritic Enteritis.—Definition.—An inflammatory disorder of the intestine, usually secondary, associated with necrosis, ulceration, and the formation of pseudomembrane. **Etiology.**—Predisposing Influences.—Chronic diseases predispose to the affection. Cancer, Bright's disease, cirrhosis of the liver may be especially mentioned. Diphtheritic enteritis is frequently a terminal infection. Exciting Causes.—Acute infections, as enteric fever and pneumonia, and certain poisons, as mercury, lead, and arsenic, are among the exciting causes.

Symptoms.—The condition may exist without symptoms. Thirst, fetor of the breath, loss of appetite, diarrhœa, ill-defined pain, dryness of the skin, and wasting are symptoms when the lesions are in the small intestine, tenesmus and diarrhœa when the lower bowel is involved. The toxic cases usually present the very acute symptoms. The clinical picture of the primary affection may completely mask the intestinal condition. Fever may or may not be present. The disease may last for many weeks. Indicanuria, increased sulphate excretion, and albuminuria are common. The fæces are not characteristic. Poor digestion of all elements is evident and the stools may be pale from undigested fat, frothy and fermenting from the starch remnants, and intensely fetid from decomposing proteids. Unaltered blood and pus may be present. Occult blood can usually be detected.

Differential Diagnosis.—*Catarrhal Enteritis.*—The symptoms in diphtheritic enteritis are more urgent. Severe pain, bloody and purulent mucoid stools suggest diphtheritic enteritis. The primary infection or the

history of chronic or acute poisoning should arouse suspicion as to the nature of the intestinal affection. *Membranous or Mucous Enterocolitis.*— Diphtheritic enteritis is an inflammatory necrosing disease with a necrotic fibrinous membrane formation. Mucous enterocolitis is a neurosis associated with but transient if any inflammation and an increased mucus formation. Diphtheritic enteritis is usually a grave disorder associated with wasting and serious symptoms; mucous enterocolitis often allows progressive increase of weight and strength. The stools of the latter condition are usually normal fecal material plus mucus. Save in an acute attack blood is not present.

Prognosis.— Occurring as a terminal infection in chronic disorders diphtheritic enteritis usually ends fatally. In the acute infections the primary disease is the important element in prognosis. The diphtheritic enteritis following the administration of poisons is always serious and often fatal.

ii. Diarrhœal Disorders of Children.

Definition.— Acute and chronic disturbances of the gastro-intestinal tract in infants associated with diarrhœa and various clinical and pathological conditions. They are usually the result of, (1) disordered digestion, (2) absorption of toxic products, (3) acute infection.

Etiology.— Predisposing Influences.—Feeble and poorly developed infants, those suffering from illness, and those nursed by ailing mothers are especially predisposed to intestinal disorders. The change from the breast to artificial feeding, particularly in the summer months, exposes the intestinal tract to infection and favors the absorption of toxins.

Exciting Causes.—Improper food, proteid or carbohydrate excess, the ingestion of milk contaminated by bacteria and their products, and, finally, a specific bacterial dysenteric infection through the milk, water, or other means, are the exciting causes. The dysentery due to the Shiga bacillus and allied organisms includes a great number of cases formerly considered due to intestinal decomposition and toxæmia—Probably the majority of the so-called summer diarrhœas.

Symptoms.— Several forms of infantile diarrhœa are recognized: (1) dietetic, (2) toxic and bacterial, (3) inflammatory, (4) chronic.

1. **Dietetic. Acute Gastro-enteritis.** — Fever, rapid pulse, anorexia, restlessness, crying, are initial phenomena. Convulsions may occur. Vomiting and diarrhœa soon ensue. The abdomen is distended, and there are evidences of abdominal pain. The vomitus is not characteristic. The stools, six to twelve or more in the twenty-four hours, are brown, watery at first, and in the mild cases becoming greenish, offensive, with green-brown mucus and fragments of undigested milk or food. In the more severe cases there is an increase of fever, together with prostration and diarrhœa and persistent offensive green acid stools. The common organisms of the intestine are always present in abundance.

2. **Toxic and Bacterial Diarrhœas.** — Infection or toxæmia may be superadded to the dietetic gastro-enteritis. More commonly the toxic and bacterial diarrhœas occur independently, following the ingestion of infected

milk, or without evident cause. *Cholera Infantum.*—The onset is abrupt and characterized by convulsions, restlessness or stupor, marked prostration, rapid wasting, and all the evidences of a severe toxæmia or infection. The temperature rises rapidly to 104°–105° F. (40°–55° C.); thirst is pronounced, and the skin is clammy and turgid, or inelastic and shrivelled in consequence of the loss of fluids by the bowel. Death may ensue in twenty-four to forty-eight hours. The bowel movements, brownish or greenish at first, but soon becoming gray and watery with abundant mucus and flocculi, are incessant. There is little or no odor and the reaction is alkaline. Blood and pus are unusual. The Shiga and allied dysentery bacilli, together with other organisms, are present in these cases.

3. **Inflammatory Diarrhœas.** — Either of the preceding forms may be followed by a localized ulcerative dysenteric ileocolitis, or the condition may be inflammatory from the start. Even though digestion and absorption may be little affected, the inflammatory and ulcerated condition of the lower ileum and colon gives rise to fever, pain, tenesmus, and frequent bloody or mucopurulent stools. Free pus or blood, usually from the lower part of the colon, may be passed. The odor of these stools is not necessarily offensive and the fecal remnants may show a fair degree of digestion. The various dysentery bacilli and the Streptococcus pyogenes are frequently found in these cases. Diphtheritic or membranous enterocolitis is a common sequel.

4. **Chronic Diarrhœa.** — Repeated attacks of dietetic diarrhœa may lead to chronic catarrhal enteritis. The inflammatory ileocolitis may persist for months. Failure to gain weight, loss of appetite, a dry wrinkled skin, nervousness, and general evidences of failing nutrition are evident. Persistent catarrh of the upper bowel is characterized by five or six loose greenish daily movements, offensive, and with excess of mucus. Undigested food is easily detected. Fermentation from the carbohydrates may be demonstrable. The putrid odor of albuminous decomposition may be recognized if the proteids are poorly digested. In chronic ileocolitis pain frequently occurs upon defecation. Mucus persists in large quantities. Blood and pus may be absent. The stools may have resumed their normal brown yellow color. Dysentery bacilli are found in some of these cases.

Diagnosis.—Usually the diagnosis is evident. Cases with persistent fever may require a Widal reaction to distinguish them from typhoid. In the chronic cases the tuberculin test may help to exclude intestinal tuberculosis. Bacteriological examination of the stools is necessary to differentiate the various forms. Serum diagnosis has not yielded positive results.

Prognosis.—In the dietetic forms with careful treatment the prognosis is good. The acute toxic and infectious cases have always a grave prognosis; only the strong infants survive. The prognosis in the inflammatory diarrhœas is likewise grave. If the infant survive the acute attack there is always the probability of an ensuing chronic ileocolitis.

In the chronic diarrhœas only exceptional cases recover, and often only after months. Many cases ultimately die of inanition or some acute terminal infection.

iii. Ulceration of the Intestines.

1. **Ulceration Restricted to the Small Intestine.**—(a) **Duodenal Ulcer; Peptic Ulcer.** — Non-malignant ulcer of the duodenum above the papilla of Vater and of the same nature as gastric ulcer.

Etiology.—Irregular habits in eating, over-indulgence in alcohol, and chronic gastritis with hyperacidity are important predisposing factors. The immediate cause is obscure. The etiology of duodenal ulcer is the same as that of gastric ulcer, and the two conditions are frequently associated.

This lesion is more common in men than in women. It may occur at any period of life, but the time of greatest frequency lies beween twenty and forty-five. It is more common than peptic ulcer of the stomach.

The ulcers may be acute or chronic. The former are circular or punched-out and more or less superficial; the latter often funnel-shaped, with thickened, indurated, and sloping borders. Duodenal ulcers are mostly near the pylorus and frequently upon the anterior wall of the gut. Gradual cicatrization with stenosis, and dilatation of the stomach, obstruction of the biliary and pancreatic ducts, hemorrhage from erosion of large arterial trunks, and perforation are common. Carcinomatous changes are very rare.

Duodenal ulcers are usually solitary; exceptionally there are two or more which present the appearance of being of different ages.

(b) **Peptic Ulcer of the Jejunum.** — This condition occurs after gastro-jejunostomy, the common site being near the point of attachment to the stomach. The causes are the same as those which lead to duodenal peptic ulcer. Hyperacidity plays an important rôle. Perforation may occur with circumscribed or general peritonitis.

Symptoms.—In both its general and local symptoms duodenal ulcer presents points of resemblance to gastric ulcer. Pain is less common in duodenal ulcer, and is apt to be late, occurring three to five hours after eating. It is referred to the right hypochondrium. Vomiting and dyspeptic symptoms are less frequent. Hæmatemesis may never occur, the blood being passed entirely by the bowel. Such hemorrhages and the resultant anæmia may be the only symptoms at first of deep ulceration. There may be an acutely localized painful area as in gastric ulcer. The test-meal shows nothing more than in gastric ulcer. Occult blood may be present in the duodenal contents removed by the method of Rehfuss.

Diagnosis.—Sudden hemorrhage from the bowel (tarry stools) or persistent evidence of blood in the stools by chemical test (occult blood), rapid or slowly progressive anæmia, pain in the right hypochondrium, occurring some hours after eating, justify the suspicion of duodenal ulcer. The anamnesis is important. Chronic recurring pain referred to the right hypochondrium, coming on several hours after eating, tarry stools, and progressive anæmia in the absence of hæmatemesis are suggestive. The diagnosis may be confirmed by röntgenography.

Differential Diagnosis.—Gastric hyperacidity with pyloric spasm and gastralgia are to be considered. The occurrence of hemorrhage, gross or occult, anæmia, and the subsequent changes due to narrowing of the duodenum are important. In gall-stone disease, jaundice, the absence of blood in the fæces, and the paroxysmal character of the pain are suggestive.

Hemorrhage and perforation are common causes of death. Each of these accidents presents characteristic symptoms of the greatest character, which do not differ in essential particulars from internal hemorrhage and perforation due to other causes. Life can only be saved by prompt surgical procedure and no time should be lost in a useless attempt to make a diagnosis.

(c) **Duodenal Ulceration Following Extensive Burns of the Skin.**—This form of intestinal ulceration occurs in about 6 per cent. of all fatal burns. The ulcers may be single or multiple, but rarely exceed six in number. The usual site is in the horizontal portion of the duodenum. They are long, narrow, and irregular in outline and commonly superficial. They have been found as early as the second day and as late as the third week, but most frequently about the end of the first week. They are more common in burns of the trunk than of the limbs, and in females than males.

Various hypotheses have been advanced to explain the restriction of this form of ulceration to the duodenum. Hunter assumes that, as a result of the impairment of function of the skin, toxic substances capable of causing ulceration of the duodenal mucous membrane are excreted with the bile. Embolism of the duodenal arteries and destruction of the antiferments in the mucous cells, with impairment of resistance to the digestive power of the gastric juice, are other explanations.

2. **Ulceration Peculiar to the Large Intestine.**—(a) Stercoral ulceration of the sigmoid flexure or colon, due to pressure of impacted fæces. (b) Dysenteric ulceration.

3. **Ulcerative Conditions Occurring in Both Small and Large Intestines** include tuberculous, malignant, syphilitic, thrombotic, embolic, and simple follicular ulceration.

The ulceration of enteric fever is fully considered in the section on the Infectious Diseases.

Etiology.—Predisposing Influences.—Any exhausting condition and old age favor the development of simple ulceration. Tuberculous or malignant ulceration may be primary or secondary.

Exciting Cause.—Pressure of the impacted fæces is the exciting cause in stercoral ulcer. The swallowing of tubercle bacilli in the sputum or food is the cause of primary tuberculous ulceration. The local determining cause of syphilitic, malignant, and many thrombotic ulcerations is unknown. Embolic ulceration follows the blocking of arterial twigs in the intestinal wall. The exciting cause of simple follicular ulceration is often an acute toxic enterocolitis, an unchecked diarrhœa, or persistent gastro-intestinal abuse. Syphilitic and tuberculous ulceration are considered in the section on the diagnosis of the specific infectious diseases.

Symptoms.—General symptoms are wasting, anæmia, thirst, and, in tuberculous and syphilitic ulceration, slight fever. Subnormal temperature is more frequent. The symptoms of an associated neoplasm or obstruction are to be considered.

A prominent symptom is persistent diarrhœa, usually painless if the ulceration is in the small intestine, attended with pain and tenesmus when it involves the large bowel. Abdominal tenderness is generally present. Mucus, pus, and visible blood may be present in large quantities when

the affection is in the colon. Thin watery movements are suggestive of small intestinal ulceration. Occult blood and excess of undigested food are usually demonstrable in the stools. Normal digestion of food substances may be but little interfered with even when the process is in the small bowel, and the examination of the fæces may fail to show excess of food remnants. Ulceration in the ileum may be unattended by diarrhœa.

Physical Signs. — A scaphoid abdomen, palpable thickening of the transverse colon, visible peristalsis, tenderness or pain on pressure, particularly along the course of the large bowel, in some cases complete absence of tympany may be observed. The physical signs of a neoplasm may coexist. Direct inspection of the sigmoid and descending colon may show the ulcerating area and is often the only positive proof of the existence of colonic ulcer.

Diagnosis.—DIRECT.—No combination of symptoms is conclusive evidence of ulceration. Detection by means of the Kelly tubes is of course final. The distinction of the various forms of ulceration may be impossible without direct inspection of the ulcerated area. Clinically the course of stercoral and follicular ulcers is usually favorable after the removal of the cause; that of malignant and tuberculous ulceration unfavorable. Stercoral ulcers are large, few in number or single, clean cut, show little or no induration or inflammatory reaction. Follicular ulcerations are apt to be numerous, small, and distinctly outlined. Considerable inflammation of surrounding tissue is usually present. Malignant ulceration is usually attended by considerable infiltration of adjacent tissues. Scrapings from the surface may show characteristic microscopical findings. Tuberculous ulceration of the lower bowel is unusual. The tuberculous ulcer is distinctly marked; tubercles may be seen. Scrapings may show tubercle bacilli, giant cells, or characteristic microscopical tubercles. Amœbæ coli are present in the fæces in the ulceration of amœbic dysentery.

DIFFERENTIAL.—*Acute and Chronic Intestinal Catarrh.*—The persistence of the symptoms, the more marked wasting, anæmia, and cachexia suggest ulceration. Direct examination by means of specula affords, in many cases, the only sure means of differentiation. The presence of small tissue fragments in the fæces is evidence of ulceration. Occult blood may be present in both conditions. *The Intestinal Neuroses.*—The more serious general symptoms, the presence of blood, mucus, and pus, and direct examination of the bowel are of diagnostic value.

Prognosis.—The outlook is usually favorable in stercoral and follicular ulceration. The course of malignant and tubercular disease of the bowel is usually uninfluenced by teatment. The prognosis in syphilitic, thrombotic, and embolic ulcerations is uncertain. The majority of such conditions are only recognized post mortem.

iv. Intestinal Stenosis and Obstruction.

Narrowing and occlusion of the lumen of the intestine due to a variety of causes, and occurring anywhere in the course of the bowel from the pylorus to the rectum.

1. **Stenosis or Incomplete Obstruction.**—**Etiology.**—Predisposing Influences.—Previous acute inflammatory conditions and malignant disease are the most important. Women, for obvious reasons, are especially predisposed to intestinal stenosis; their greater liability to enteroptosis also increases their liability to stenosis from the kinking or twisting of misplaced intestines.

Exciting Causes.—The direct causes are many and may be enumerated according to the location of the narrowing. In the small intestine the cicatrices of duodenal ulcers, gall-bladder and common bile-duct disease, diseases of the head of the pancreas, cancer of the duodenum, jejunum, or ileum, omental and peritoneal adhesions, and accidents, as hernia, adhesions or compression caused by new growths, and involvement of the gut in inflammatory diseases of the appendix or pelvic organs are the most common causes. In the large intestine peritoneal and appendicular adhesion, adhesions to the gall-bladder, adhesions to and compression by pelvic tumors are common causes of stenosis. Cicatricial narrowing following dysenteric and stercoraceous ulceration is an occasional cause. Tuberculous ulceration of the large bowel is less often followed by stenosis, while syphilis of the rectum, with ulceration and resulting narrowing, is a frequent cause. Cancerous invasion of the rectum and sigmoid flexure is one of the most common causes of intestinal stenosis.

Symptoms.—General.—General symptoms in intestinal stenosis may depend upon the cause more than upon the narrowing of the bowel. Anæmia, wasting, and loss of appetite occur early in cancerous stricture and in tuberculous and syphilitic disease, whether the stenosis is of extreme grade or not. In the mechanical stenosis due to pressure and adhesions, unless they are near enough to the stomach to cause early dilatation and vomiting, wasting may not occur and anæmia may be long absent. Mental and physical depression are usually marked in persistent stenosis. Thirst is a common symptom; oliguria occurs.

Local Symptoms.—The situation is important. The fluid contents of the upper bowel may be easily forced through an opening which would be occluded or readily obstructed by the solid fæces of the lower bowel. Stenosis above the ileum sooner or later produces a dilatation of the stomach with marked local gastric symptoms. Stenosis in the colon is likely to be attended by constipation, a symptom not common in narrowing at a higher level. Finally, tenesmus and intensely painful muscular contractions are limited to stenoses of the large bowel.

Stenosis of the Duodenum and Jejunum.—Distention after eating, eructations, gradually increasing and persistent nausea, and finally vomiting are common local symptoms. Persistent biliary vomiting suggests stenosis below the papilla of Vater, while symptoms of disturbed hepatic and pancreatic function occur when the common and pancreatic ducts are involved in the lesion causing obstruction. Painful contractions of the intestine are not usual in stenosis of the upper parts of the small intestine. The vomitus is that of gastric dilatation; it is not fecal. Bile is present under the conditions just mentioned. Pancreatic ferments may be recognized. Blood tests may be positive if ulceration exists. The fæces are not distinctive. Associated occlusion of the bile and pancreatic

ducts may show unabsorbed fat and undigested proteids, and carbohydrates in excess. Blood tests will be positive if there is ulceration.

Stenosis of the Ileum.—Distention following the intake of food is less common. The stomach is not usually dilated. Nausea and vomiting of gastric and intestinal contents occur, but not continuously. Stenosis situated low in the ileum may be associated with a slightly fecal smelling vomitus. Painful contractions of the bowel do not often occur, but in some cases persistent crampy pain with moderate distention is the only symptom. Many ileal stenoses never reveal themselves till a sudden occlusion produces an acute obstruction.

Stenosis of the Large Bowel.—Distention of extreme degree may be present, particularly if the stenosis is very low. Constipation is the rule, or constipation alternating with diarrhœa. Vomiting occurs occasionally, but is only fecal when it has persisted for some hours and acute obstructive symptoms have supervened. Tenesmus and painful muscular contractions of a periodic type are characteristic of lower bowel stenosis. With ulceration blood is present in the fæces. There is, however, no characteristic stool in stenosis of the lower bowel.

Physical Signs.—Inspection.—The existence of anæmia and cachexia, and wasting are to be noted. Marked distention is often apparent in stenosis of the large bowel. Stenosis of the small bowel is less apt to give rise to extreme distention. A distended stomach may be apparent in cases of duodenal stenosis. Inspection frequently shows a tense intestinal tube (intestinal rigidity) or several, one above the other (ladder pattern), and these rigid distended parts may further show energetic peristaltic movements running up to and ending in the obstruction and sometimes bringing a stenosing tumor into view. The colon or its sigmoid flexure may be clearly outlined. The latter may occupy the whole abdomen. Inspection may also reveal tumors, a protruding hernia, fulness in the hernial tracts, and the scars of abdominal operations which suggest adhesions or constricting bands. Palpation.—The rigidity of an intestinal tube above a stenosis is easily appreciated. Peristalsis with muscular hypertrophy and a stenosing tumor of the bowel may be felt. Abdominal tumor, hernia, adherent scars, and the like can be readily examined. Percussion is of limited value. Auscultation, beyond allowing us to hear fluids trickling through an aperture and to conclude that it is still patent, does not afford any aid in diagnosis.

Inflation of the stomach in the endeavor to determine duodenal stenosis gives no clear result. Inflation of the large bowel may, in thin subjects, reveal a stenosis in the upper part of the sigmoid flexure. Stenosis of the descending colon or transverse colon may become evident, but as a general rule the natural distention above the stenosis is more distinctive. Rectal examination in intestinal stenosis may reveal the occluding mass of a pelvic tumor, narrowing of the anus and lower rectum due to stricture, or the rough, hardened, ulcerating edges of a malignant growth. Vaginal examination may at times reveal palpable tumors or stenosing conditions in the pelvis or adjoining intestines. Proctoscopic and sigmoidoscopic examination will show the presence of stenosing conditions, cicatrizing ulcers, syphilitic or fibroid stricture, or the narrowing of the

intestinal tube from outside pressure. The X-rays after barium injection or ingestion may yield important diagnostic facts. A well-outlined sigmoid or colon may show acute kinking, a tumor, or constriction preventing the passage of the barium beyond a certain point even after many hours. Equally accurate information may be obtained in stenosis of the small intestine.

Diagnosis.—Direct.—Distention, abdominal pain, cramp-colic, tenesmus, constipation, or alternating diarrhœa suggest stenosis of the large bowel. Persistent gastric symptoms and gastrectasis, recurrent vomiting with no evidence of pyloric tumor or pyloric obstruction, and continuous biliary vomiting direct attention to the small bowel as the seat of trouble. The recognition of an intestinal tumor, the appearance of intestinal rigidity, intestinal patterns, visible peristalsis, and visible and palpable muscular hypertrophy make the diagnosis sure. Fecal vomiting, which usually indicates that the condition has passed from stenosis to complete obstruction, localizes the obstruction a very short distance either above or below the ileocæcal valve.

Differential.—*Nervous Dyspepsia, Nervous Flatulence and Vomiting, Enteralgia.*—Gastric dilatation as seen in duodenal stenosis, intestinal rigidity, intestinal patterns, and visible and palpable intestinal peristalsis do not occur in the neuroses. Vomiting in the neuroses is easy, is apt to occur immediately after eating, and the vomitus is usually undigested, odorless food. General symptoms with the exception of wasting are less marked. *Lead Colic.*—The diagnosis may be impossible, since temporary stenosis undoubtedly takes place during the spasmodic contraction. Marked anæmia, with the early symptoms of intestinal disorder, and a blue line on the gums may be the only distinguishing features. *Persistent Vomiting of Alcoholism, Locomotor Ataxia, Gastroxynsis.*—In these conditions the general symptoms are practically absent. The history is different, and in tabes characteristic ocular and nervous phenomena are present. The vomitus in these conditions consists of mucus and a watery gastric secretion, and never contains food remnants or has a fecal odor, no matter how persistent and profuse it may be. *Distention of the Intestines (Paretic Distention) of Acute Fever; Idiopathic Dilatation of the Colon.*—In these conditions stenosis, pain, colic, complete constipation, and vomiting are absent, though faint visible peristaltic and intestinal patterns may sometimes be seen. Obstruction hypertrophy and palpable muscular contraction do not occur.

Prognosis.—The prognosis depends upon the cause of the stenosis. Cancerous stenosis, unless a diagnosis has been made in time to allow resection, is fatal. Any simple stenosis may terminate suddenly in a fatal obstruction, but the majority can be relieved by operation.

2. **Complete Obstruction.**—Many chronic stenoses end in acute obstruction. Various accidents, strangulation, twistings of the bowel, volvulus, etc., produce the same results—complete occlusion of the bowel, the retention of flatus and intestinal contents, and the sudden development of serious symptoms.

Etiology.—Predisposing Causes.—*Chronic Stenosis.*—Bands of adhesion and open hernial canals constitute predisposing factors.

Exciting Causes.—Excesses at table or accumulation of the residua of coarse food or of other material may suddenly block the narrowed gut. Twisting of the bowel, various hernial accidents, and intussusception are immediate causes. The settling of a large pelvic tumor is a not uncommon direct cause of acute obstruction. Foreign bodies, gall-stones, accumulation of parasites, and impaction of fæces are also direct causes. Almost any acute abdominal inflammation may give rise to acute obstruction.

Symptoms.—The general symptoms are marked and severe. In simple stenosis, so long as a narrowed opening remains patent, general symptoms may be absent. Large collections of fecal material may accumulate without causing marked discomfort, but the moment the obstruction becomes complete serious symptoms supervene. Rise in the pulse-rate, increased vascular tension, slight elevation of temperature followed by the signs of prostration and collapse, pallor, sweating, facies abdominalis, thready pulse, and shallow respiration constitute a symptom-complex at once alarming and significant. Thirst and oliguria are constant. Intense grinding pain, eructations, nausea, and persistent vomiting, at first of gastric juice, later of gastric fluid commingled with bile or intestinal contents and bile, and, when the obstruction is below the ileocæcal valve, of distinctly fecal material, speedily occur. The vomitus in obstruction of the lower ileum may have a faintly fecal odor. Distention is a constant phenomenon and is more marked the lower the obstruction. Volvulus and intussusception may be accompanied by the passage of blood, and in sigmoid obstruction tenesmus with bloody mucous discharges occur. Neither fæces nor flatus are passed per rectum. In unrelieved cases peritonitis rapidly develops with intestinal paresis, generalized pain, and marked meteorism.

Diagnosis. — Direct. — Grave general symptoms, abdominal pain with intense exacerbations, tenesmus, nausea, eructations, persistent vomiting eventually becoming of a fecal character, absolute failure to pass flatus and fæces, distention, intestinal patterns, rigidity, and tumefaction of the intestine as the contraction reaches the obstruction are the main diagnostic features. Changes in the urine such as the appearance of excess of sulphates (indican) are not diagnostic. Leucocytosis is not constant and therefore not an important diagnostic feature.

Differential.—There are but few conditions which simulate acute obstruction. Thrombosis of the mesenteric vessels, acute pancreatitis, and acute enteritis with relaxation of the intestinal coils, pain, and vomiting, may be mentioned. The absence of intestinal patterns, rigidity, and visible and palpable peristalsis are of value in differentiating these conditions from acute obstruction in cases seen prior to the development of peritonitis and paralytic distention. Acute appendicitis with peritonitis may closely resemble acute obstruction. A history of attacks of pain in the region of the appendix is often obtained. The diagnosis may be difficult. Spontaneous relief is rare. Early surgical intervention is imperative. In neglected cases death ensues in the course of three to six days.

Obstruction of the large bowel from accumulation of hardened fæces is rarely complete. Fecal obstruction can be differentiated from stenosis due to other causes by the recognition of the accumulated masses, the

comparative mildness of the general and local symptoms, the absence of marked visible and palpable intestinal peristalsis, and the relief afforded by judicious therapeutic measures.

In the investigation of a case of acute intestinal obstruction the object is to determine the nature and cause of the stoppage and its location.

The obstruction may be due to mechanical causes, as collections of scybala, enteroliths, masses of intestinal worms, large biliary calculi, foreign bodies that have been swallowed, new growths in the intestinal walls, contracting cicatrices following ulceration or surgical operations, or the pressure of tumors. Under these circumstances the primary fault is not in the wall of the gut and the condition is described as *simple occlusion*.

When the obstruction arises in consequence of an injury to a segment of the intestine and its mesentery by which they are deprived of their blood supply, the condition is known as *strangulation*. This may occur in incarceration of external or internal herniæ, volvulus, intussusception or from the action of bands or adhesions following previous abdominal operations in consequence of changes in the contents of the gut, or unusual peristaltic movements.

Strangulation is rapidly followed by necrosis.

In many cases the location may be determined by a physical examination, or röntgenography, but frequently prompt surgical operation is imperative. In every case of acute obstruction, a surgeon should be at once called to watch the course of the symptoms and food; opiates and especially purgatives must be withheld pending the decision as to the proper course of treatment.

Forms of acute obstruction not directly caused by mechanical conditions are known as paralytic. They most frequently arise in consequence of acute inflammation of the peritoneum following perforating ulcers, the rupture of cysts or abscesses, traumatism, or abdominal operations. Not rarely they are toxic in origin, occurring in the course of severe infections, as enteric fever, variola or pneumonia or they may occur in sepsis or uræmia. Exceptionally this form of acute intestinal obstruction appears to be reflex, as when it suddenly develops during an attack of biliary or renal colic or in injury to or inflammation of a testicle. The obstruction is not due to obturation but to loss of the peristaltic function of the bowel. There is enormous tympanitic distension of the abdomen, silence on auscultation, no great tenderness, and nearly complete absence of recurrent spasmodic pains.

v. Dilatation of the Intestines—Idiopathic Dilatation of the Colon.

Definition.— Chronic dilatation of the colon and sigmoid flexure, not due to stricture or accumulation of fæces.

Etiology.—Predisposing causes are unknown. A history of chronic constipation is usually obtained. The condition usually occurs in quite

young persons and children. The pseudocyesis of middle-aged women depends largely upon dilatation of the colon and sigmoid flexure.

Exciting Causes.—Fecal accumulation plus paretic distention of the bowel occurring repeatedly produces the condition. Spasmodic contraction of the rectum must coexist. Idiopathic dilatation due to structural abnormalities is a probable cause.

Symptoms.—The general health may be but little affected. Acute symptoms rarely occur. Extreme distention gives rise to respiratory and cardiac oppression. Constipation is the rule, but diarrhœa occurs. The most prominent symptom is distention. Pain, colic, and obstructive symptoms are absent. The distress that accompanies distention of the small intestine is not observed in idiopathic dilatation of the colon. Enormous distention of the abdomen, thinning of the abdominal walls, the presence of lineæ atropicæ, and glazing of the skin are seen. With marked wasting of the abdominal walls the outline of the sigmoid flexure rising from the pelvis and reaching to the costal margin, or the outline of the colon, can be made out. Peristalsis is not pronounced. Palpation shows the distention to be gaseous. No resistance or solidity is felt; no fluctuation wave obtained. Percussion gives a marked tympany everywhere, even in the loins up to the base of the lungs behind, and often shows an obliteration of the liver dulness in front. The passage of a soft rubber tube into the sigmoid flexure relieves distention by allowing the exit of air and shows what part of the bowel is affected. Reinflation through the tube produces a gradual ballooning and outlining of the sigmoid or colon. Examination by means of the speculum shows merely the relaxed condition of the colon when the air is expelled.

Diagnosis.—Direct.—The absence of serious local and general symptoms, constipation, extreme chronic distention with outlining of the sigmoid or parts of the colon, the disappearance of distention on passing the rectal tube, and the results of inflation are characteristic.

Differential.—The general symptoms and the relief of the distention by the tube differentiate dilatation of the colon from *gastrectasis*. The shape and position of the stomach are radically different. *Distention of the Small Intestine due to Obstruction or Paretic Conditions of the Musculature.*—The general symptoms of disease of the small intestine are more marked. Intestinal patterns are smaller and more numerous; peristalsis (save in paretic distention) may be seen. *Distention of the Large Bowel due to Stricture, Malignant Growth, etc.*—The distention due to obstruction is associated with signs of associated muscular hypertrophy. Visible or palpable active peristalsis, pain, and colic are common. General symptoms rapidly develop. The obstruction can often be recognized by the speculum or examining finger, or upon palpation through the abdominal wall. *Fluid Accumulations.*—Bulging of the flanks, movable dulness on turning, fluctuation wave, flatness on percussion readily distinguish fluid accumulation from gaseous distention.

Prognosis.—The disease in itself is rarely fatal. In the idiopathic cases death commonly occurs early in life. General treatment has little

effect. Removal of the distended sections of the colon and sigmoid flexure has been practised.

In diffuse dilatation of the colon there is a tendency to fecal accumulation with troublesome constipation. Fermentative processes occur and result in an absorption of toxic substances—*intestinal autointoxication.* Upon theoretical considerations many constitutional symptoms have been attributed to this form of toxæmia. Among them are headache, lassitude, sallowness, biliousness, loss of flesh, nervousness, neurasthenic and psychasthenic symptoms. Lane made a study of the subject (1910–1913) and described a variety of deformities in addition to colonic dilatation, as Lane's kinks, etc., which interfere with intestinal drainage. He devised a number of operations for the relief of these conditions, release of kinks, forms of intestinal anastomosis, extirpation of the colon, and so on. Remarkable results were attributed to this work for a time. But the enthusiasm it aroused shortly subsided and the whole subject has taken its proper place among surgical procedures.

vi. Diverticulitis.

Definition.—Inflammation of intestinal diverticula. Clinically the diverticula of the descending colon and sigmoid are involved.

Etiology.—Predisposing Influences.—Age plays an important part, since diverticula are infrequent in young persons and do not form conspicuous alterations in the walls of the gut until about the fiftieth year or later. They develop in fat persons or those who have been fat. Males are much more frequently the subjects of diverticulitis than females. The internal pressure upon the wall of the colon in habitual constipation and flatulent distension are contributory causes in the formation of diverticula and their distension with fecal matter. Their common position is close to the attachment of the mesocolon.

The Exciting Cause.—The inflammation is the result of bacterial infection and may be acute or chronic. The acute form may be catarrhal or suppurative with or without abscess formation. The chronic form is commonly proliferative and adhesive.

Symptoms.—Pain, tenderness, constipation, muscular rigidity and a palpable tumor in the left iliac fossa are present. Abscess formation may occur with obscure fluctuation. Perforative peritonitis may result. The condition in acute cases closely resembles appendicitis except that it is upon left side. Fever, and a polymorphonuclear leucocytosis may be present. The occurrence of chills suggests suppuration. In the chronic forms a tumor mass may simulate carcinoma.

Diagnosis.—Direct.—This depends, in the acute cases, upon the concurrence of the above clinical manifestations in a person above forty years of age, especially if, in the anamnesis, constipation is prominent. In the chronic cases the röntgenological examination is conclusive, the barium remaining in the diverticula after it has disappeared from the colon.

Differential.—There may be difficulty in differentiating the acute form from a left-sided appendicitis. The focus of tenderness in the latter is

not so deep in the iliac fossa; vomiting is more apt to occur; a rectal examination should be made. Specific lesions of the colon due to tuberculosis, syphilis, actinomycosis, and dysentery yield characteristic diagnostic phenomena and are relatively infrequent. Inflammatory disease of the pelvic organs require the methods of gynæcology. Röntgenology may be necessary in any case.

Prognosis.—Remarkable improvement takes place in many cases under judicious medical management. In others prompt and efficient surgical measures may be necessary to save life.

vii. Appendicitis.

Definition.—Inflammation of the vermiform appendix.

The conception of appendicitis is a modern one, dating from the studies of Reginald Fitz (1886). It includes and explains the facts relating to foreign bodies in the appendix; catarrhal, diffuse, purulent, and necrotic inflammation of that organ; ulceration, gangrene, cyst formation, and abscess of the appendix; chronic, recurrent, and obliterative inflammation; peri-appendicular abscess, typhlitis, perityphlitis, and iliac phlegmon; and local and general peritonitis having its starting-point in the ileocæcal region.

These conditions constitute phases in the evolution of a single process—appendicitis. The central fact is infection of the appendix.

The infection may be a local manifestation of a general infection, as, for example, influenza or pneumonia: or a purely local process, as in the case of foreign bodies, fecal concretions, the extensions of an inflammation from the cæcum, or when injury to the appendix results from strains or blows; and finally the infection may be associated with a specific local lesion in a general disease, as when typhoid ulceration involves the lymph tissue in the appendix. Kelynak (1903) suggested that acute appendicitis is a metastatic inflammation arising from a distant primary focus of infection. Other observers, notably Apolant and Kretz, have recently advanced the opinion that "appendicitis begins as a metastatic disease of the adenoid tissue, and that the lymphatic apparatus of the throat and nose is to be regarded as the most frequent primary localization and portal of entry of the infection." The recognition of the unity of the process under varying etiological conditions and in varying clinicopathological manifestations is the key to its diagnosis and treatment.

Etiology.—Predisposing Influences.—Appendicitis is the most important and one of the most common of the acute diseases of the intestine. There are no especial causal relations connected with country, race, social conditions, or occupation, save that it has been held that those whose occupations involve habitual strain and heavy lifting suffer more commonly than others. About half the cases occur before the twentieth year. It is rare in infancy but common in childhood and adolescence. Cases have been observed as late as the seventh and eighth decades. It is equally common in the two sexes. The symptoms have occasionally followed a

fall or blow upon the abdomen. Indiscretion in diet, especially overeating, and exposure to cold and fatigue are conditions frequently mentioned in the anamnesis. The acute infections, in particular influenza, pneumonia, and rheumatic fever, sometimes are attended with or followed by appendicitis. A majority of the cases, however, arise in ordinary health without any obvious or discernible determining cause. Two or three cases in the same house at or about the same time have occasionally been observed.

The Exciting Cause.—Various pyogenic organisms have been found in the early lesions, among which Bacterium coli communis and Streptococcus pyogenes are common. A lesion of the mucosa, caused by the presence of a foreign body, fecal concretions, retained secretions, or traumatism, probably constitutes the point of entrance for pathogenic bacteria.

Nature of the Pathological Process.—The character of the lesions is determined by the intensity of the infection and the reaction of the tissues involved. Broadly speaking, the lapse of time between the onset of the attack and the condition at any given hour has a most important bearing upon the anatomical diagnosis; that is to say, the early lesions are relatively simple and limited, the later complex and extensive. But to this rule there are many exceptions. In a large group of cases, the so-called catarrhal cases, the inflammation runs a favorable course, resolution takes place in a short time, and in a few days the patient is convalescent. But the recovery is by no means always complete in the sense of an anatomical *restitutio ad integrum.* The inflammation subsides but the appendix remains infected and lesions of a chronic and progressive nature persist—infiltrations of the mucosa and submucosa, connective-tissue overgrowth, local atrophies involving especially the longitudinal and circular muscular fibres, stricture-like narrowings, retained secretions, cyst formation, angular kinking, adhesions, and other deformities. It is in such cases that the teasing pains known as appendicular colic occur, in which there is persistent discomfort and frequent tenderness in the right lower quadrant, in which inflammatory flare-ups recur, and in which at any time necrosis, perforation, abscess formation, or general peritonitis may suddenly arise. They constitute the cases of so-called recurrent appendicitis, an unfortunate and misleading term since this form of the disease is in point of fact essentially chronic with occasional exacerbations—a smouldering fire with now and then an ominous puff of flame. On the other hand there are cases in which the symptoms of onset are urgent, and necrosis and perforation follow in the course of a few hours. Many of the cases, however, make a substantive recovery and live on without subsequent attacks. Others, which constitute a large proportion of all cases, go on more or less rapidly from bad to worse, terminating in abscess formation and chronic invalidism, or general peritonitis and death.

The natural history of appendicitis is indicated in the following table.

The course of the attack may be interrupted and in a majority of the cases its more serious events and unfavorable terminations arrested by early surgical intervention.

Symptoms.—The significance of the clinical phenomena becomes more apparent from a careful consideration of the following pathological data:

APPENDICITIS.

I. Acute.

Pathological variety.	Onset.	Course.	Termination.
A. Catarrhal	Abrupt	Brief; a few days	(a) Recovery without subsequent attacks. (b) Recovery with subsequent attacks at long intervals. (c) Chronic forms: (1) Apparent recovery, with subsequent attacks at irregular short intervals, with uneasiness and pain in the intervening periods. (2) Recovery from acute symptoms with persistent local pain and tenderness, intestinal symptoms, and other indications of ill health. (3) Chronic thickening and hyperæmia, without symptoms. (4) Thickening with fibrotic changes, an involution process; the obliterative form. (d) Suppuration.
B. Suppurative	Abrupt. May follow the catarrhal form	Variable; 24 to 36 hours, several days	(a) Pus formation: when the lumen is obliterated the distal appendix may be converted into an abscess cavity; more commonly necrosis occurs. (b) Local peritoneal infection without actual perforation; local adhesive peritonitis and peri-appendicular abscess. (c) Necrosis.
C. Ulcerative	Gradual in the course of the catarrhal form from pressure of a foreign body or appendolith, or in the suppurative form from intense local action of pyogenic organisms	Variable; cannot be known	(a) Peri-appendicular adhesive inflammation. (b) Perforation and abscess formation. (c) Perforation and peritonitis.
D. Gangrenous	Abrupt, or may follow suppurative form, or be fulminant	Very variable; may occur after several days of suppuration or ulceration, or with urgent, even fatal, symptoms in the course of a few hours	(a) Appendicular abscess, circumscribed or extensive. (b) General peritonitis, rarely adhesive; commonly ichorous or purulent, and almost always fatal. (c) When necrosis occurs at the site of adhesions to neighboring organs, as the gut or bladder, a fistulous communication is established through which pus is discharged. (d) Hemorrhage.

II. Chronic.

E. Primary fibrosis	Insidious	Prolonged; indefinite without marked clinical manifestations	(a) Thickening of appendix with progressive obliteration of lumen beginning at distal extremity. (b) General atrophy and sclerosis.
Secondary fibrosis	Obscure. May be insidious or a terminal condition following an acute form	Prolonged; condition may not be suspected during life	(a) Thickening and enlargement; elongation or kinking; narrowing of lumen. (b) Chronic adhesive inflammation to adjacent structures with thickening and signs of solid tumor.

Catarrhal inflammation of the appendix may be acute, subacute, or chronic. The mucosa is slightly involved, being swollen, injected, and the seat of an increased secretion. Inflammatory thickening may cause retention of the secretion and persistence of the infection. In the subacute form symptoms may be absent. In the acute form they are of variable intensity, consisting of pain and tenderness in the right lower quadrant of the abdomen, together with loss of appetite, nausea, and occasional fever, which is usually of slight intensity and brief duration. In the chronic form there is persistent tenderness with a sense of thickening upon deep palpation, vague and often intractable gastro-intestinal symptoms, neurasthenia, and in some instances an associated mucous colitis.

Acute diffuse inflammation is more common. The infection extends from the mucosa to the deeper structures. There is inflammatory thickening and hyperæmia which involves the serous investment. Erosion and ulceration of the mucosa are common. The symptoms are more severe than in the acute form and the duration of the attack is prolonged.

Acute suppurative inflammation may supervene. There may be purulent infiltration of the wall of the organ, or the lumen may be occluded so that the pus distal to the stricture forms an abscess cavity. The symptoms are now more severe and a mass may be felt through the abdominal wall. An extension of the purulent infection or a small perforation may be followed by peri-appendicular suppuration and cause a distinct fluctuating tumor. Septic phenomena are often present, as irregular fever, sweating, rapidly developing anæmia, and gastro-intestinal symptoms, especially nausea and vomiting. In many of the cases, however, these symptoms do not occur. Fever in particular may be wholly absent.

Ulceration may follow catarrhal or diffuse inflammation and cause more or less extensive subacute local peritonitis with adhesions. More rarely it may cause perforation with abscess formation. The lesion advances slowly and is far less likely than gangrene to cause general peritonitis. There are no special symptoms of ulceration other than those due to the subacute circumscribed adhesive peritonitis which it causes, namely, local pain, tenderness, rigidity, and a more or less well-defined tumor mass.

Gangrene is common. It may occur in the course of a few hours after the symptoms of onset. In a case in which pain was first felt after the bath at eight in the morning, the whole appendix was found necrotic upon operation at five in the afternoon. Instances of this kind are by no means exceptional. More commonly gangrene takes place, in unrecognized cases or those in which operation is refused, in two or three days or later.

The necrotic process may involve the tip, or a circumscribed patch, or the entire organ, or it may be confined to the region of its attachment to the cæcum. The appendix may slough off and be found loose in the pus of an appendicular abscess. When the necrotic tip has previously become adherent to a neighboring hollow organ, as the colon or bladder, a fistulous communication may be found. The appendix has in some instances perforated into a hernial sac. The necrosis has occasionally involved the wall of an artery, as one of the iliacs, to which adhesions have formed, and led to fatal hemorrhage. Perforation, which only

occasionally occurs in the acute diffuse and suppurative forms, is the usual outcome of the gangrenous variety. The opening is commonly single, but multiple perforations may occur. When previously formed adhesions confine the escaping contents of the appendix, an abscess is found which varies in size and may, in the absence of operation, ultimately discharge into the bowel or bladder, vagina, or externally.

Chronic appendicitis constitutes a common condition when the acute symptoms subside without operation. The appendix may be thickened and deformed, adherent to the gut, or embedded in a mass of irregularly puckered peritoneal adhesions. Its lumen may be more or less completely obliterated, or it may be converted into a series of cysts separated by stricture-like occlusions, or, finally, it may contain foreign bodies or fecal concretions. There are cases in which no symptoms are present, but it is more common to find occasional pain, persistent tenderness, and intestinal symptoms. When acute exacerbations of these symptoms occur the condition is known as recurrent appendicitis, falsely so called.

In another group of cases chronic appendicitis takes the form of a progressive primary fibrosis, a progressive involution process, not characterized by symptoms and terminating in complete atrophy of the organ.

The bodies found in the lumen of the inflamed appendix are, as a rule, to which there are few exceptions, fecal in character. The pus varies in amount and consistence. It may be soft and easily expressed, or consist of dense, compact masses resembling date-stones or grape seeds. A series of three or even more such masses may occupy the canal of the appendix. Sometimes there is only one, which is short and rounded, or elongated and curved. Of foreign bodies the most common are pins.

The symptom-complex of acute appendicitis is most variable. Certain symptoms are, however, suggestive, their association significant. The chief of these are pain, tenderness, and gastro-intestinal disturbance. Of subordinate importance are fever, muscular rigidity, deep induration, and leucocytosis. We cannot be satisfied with an affirmative reply to the question: Has this patient appendicitis? We must ask ourselves further: What stage in its evolution has the process reached? Is the appendix alone affected? Are there signs of abscess formation? Is there local adhesive peritonitis without or with pus? Are there signs of a beginning general peritonitis? In other words, What are the lesions?

1. Pain.—The pain is usually sudden, mostly severe. It is sometimes sharp and stitch-like; sometimes dull and aching. It is often colicky; almost always continuous with exacerbations. It is usually referred to the right lower quadrant of the abdomen, but may be diffuse or central. In the chronic cases a diffuse central pain is very often present for weeks or months before the pain in the right iliac fossa is felt. It may be referred to the region of the gall-bladder or to the right lumbar region, or extend in these directions; or it may extend in a similar way to the perineum or right testicle. Under these circumstances the pain of appendicitis has been mistaken for biliary or renal colic. These referred pains have been ascribed to the position of the appendix, which has been found in some instances elongated in the direction of the liver or kidney, in others downwards into the pelvis.

2. Tenderness.—Firm, deep pressure over the affected area usually causes pain. A circumscribed spot situated at the intersection of a line drawn from the umbilicus to the anterior superior spine of the ilium and a second line corresponding to the outer border of the right rectus muscle, first described by McBurney and known as McBurney's point, is in well-marked cases decidedly, even exquisitely, tender to the pressure of a single finger. Deep pressure with the open hand upon the left side of the abdomen frequently causes or aggravates the pain in the right iliac fossa. The patients usually assume the dorsal decubitus with the right knee flexed, and pain is elicited or increased by extending the limb.

3. Gastro-intestinal Symptoms. — There are loss of appetite, thirst, nausea, and very often vomiting which ceases in the course of some hours. Constipation is the rule. Diarrhœa occasionally occurs in children. The patients are often able to recall the eating of an unusually hearty meal or some indigestible article of diet, or undue exertion or exposure after food, and attribute pain and tenderness to indigestion or colic or a bilious attack.

4. Fever.—An initial chill or chilliness is very rare. It is said that fever is always present at the onset. If so, it is very often transient and has disappeared before the case comes under medical observation. Frequently the temperature is normal when the patient is first seen, and in favorable cases remains so. Many cases show subfebrile temperatures not reaching 101° F. (38.5° C.), and fever of irregular type—102°–3° F.—is not uncommon. There are cases in which, with abscess formation or general peritonitis, the temperature remains low, and others in which fever gradually subsides and the patient enters upon convalescence without serious symptoms. Too great reliance upon the thermometer may prove misleading.

5. Muscular Rigidity.—Spastic tension of the abdominal wall upon the right side, especially over the rectus muscle, is common in severe cases. In many cases this symptom is not present. Its presence is suggestive of a beginning local peritonitis.

6. Induration.—Upon deep palpation a circumscribed sensation of resistance may frequently be detected. This sign is often well defined and situated at or near McBurney's point. In very rare instances the thickened appendix may be distinctly recognized upon palpation. In other cases there is a diffuse ill-defined boggyness with some impairment of resonance upon percussion. Upon the supervention of pus the signs of a solid or fluctuating tumor are characteristic. Necrosis and perforation may take place without either induration or a palpable tumor, especially in the early gangrenous cases.

7. Leucocytosis.—In appendicitis unaccompanied by suppuration, gangrene, or serous inflammation, there is usually little increase in the leucocytes. In cases which have gone on to abscess formation, necrosis, and local or general peritonitis, leucocyte counts of 15,000 or more per cubic millimetre are the rule. Normal counts or slight leucocytosis may occur in cases characterized by early gangrene and perforation,—so-called fulminant appendicitis,—and in suppurative cases in which absorption does not take place from the abscess cavity.

8. Inconstant and therefore unimportant symptoms are vesical irritability, oliguria, and albuminuria. As in many other acute inflammatory affections, there is moderate early anæmia of secondary type. In cases attended with prolonged suppuration, with sepsis, anæmia becomes pronounced with a reduction of hæmoglobin to 30 or 40 per cent. and an erythrocyte count of 3,000,000 per cubic millimetre or less.

The course of a case of simple appendicitis terminating favorably is usually as follows: The gastro-intestinal symptoms subside, the tongue cleans, and the constipation ceases spontaneously. The appetite and strength return. The pain gradually disappears, but tenderness outlasts it for some days or a week or two. Local induration progressively diminishes and convalescence is soon fully established. The persistence of a distinct circumscribed tumor is very liable to be followed by recurrent outbreaks.

Abscess and peritonitis are sequels of appendicitis.

Abscess.—The conditions revealed at operation shed much light upon the development and course of appendicular abscess. Perforation either as the result of ulceration or necrosis is the common cause of peri-appendicular suppuration. In exceptional cases it occurs in consequence of general suppurative inflammation of the appendix. Pus may be found in the course of two or three days, but more commonly towards the close of a week. The symptoms are aggravated. There are more or less pronounced signs of sepsis and fever of irregular type. There are, however, cases in which fever is wholly absent. The pain and tenderness are often increased. The abscess cavity is usually in relation with the appendix and the adjacent coils of intestine, which are adherent among themselves as the result of local plastic peritonitis. In some of the recurrent cases there are one or more small collections of pus in an irregular mass formed by dense adhesions and puckering of the wall of the gut. More commonly the abscess cavity is single and its size corresponds to the duration of the case. When small and deeply seated, especially if below the pelvic line, it may elude external palpation but be detected upon vaginal examination. As a rule, it forms a palpable or visible tumor of variable size in the right iliac fossa, which in some cases is distinctly fluctuating. The pus shows the usual tendency to burrow and may discharge into the bowel, vagina, or bladder, or externally.

Peritonitis.—General infection of the peritoneum may at once result from early necrosis, prior to the formation of circumscribing adhesions, or later from the rupture of already formed adhesions. In the fulminant cases the symptoms of general peritonitis may follow those of appendicitis so rapidly that it is not always possible to determine the nature of the primary process. Ordinarily the onset of peritonitis is attended by sudden aggravation of the previous symptoms. The pain, tenderness, and rigidity become more marked and extend over the entire abdomen. Nausea and vomiting are more severe. The pulse becomes small and rapid, the tongue dry, and the urine scanty or suppressed. After a time meteorism, absence of peristaltic movement, dorsal decubitus with flexed thighs, and the characteristic facies complete a well recognized clinical picture. Cessation of pain and a clear mind are the heralds of death.

Diagnosis.—Direct.—The recognition of acute appendicitis rests upon the association of sudden pain and tenderness in the right iliac region with nausea or vomiting. The presence of a circumscribed tumor or deep resistance and rigidity of the right rectus muscle are significant. The age of the patient is suggestive, since appendicitis is very common before and comparatively rare after thirty. Conditions having some resemblance to appendicitis are to be carefully excluded, as, for example, hepatic and renal colic, dysmenorrhœa, and tubo-ovarian disease. The diagnostic importance of the blood counting may be readily overestimated.

Differential.—1. *Cholecystitis.*—When the inflamed appendix lies upwards, the pain and tenderness may suggest gall-bladder disease. In the latter careful physical examination will usually reveal increased dulness and circumscribed tenderness in the region of the gall-bladder, and a history of recurrent attacks without tendency to inflammatory tumor or abscess formation. 2. *Renal Colic.*—It is only in the rare cases in which the appendix extends backwards that uncertainty arises. The pain of renal colic is usually more severe and more distinctly paroxysmal than that of appendicitis. It arises in the lumbar region, extends forward and downward towards the groin, and is attended by retraction of the testicle. The diagnosis in some cases of persistent kidney colic with hydronephrosis is very difficult. The X-ray examination may be of service. Dietl's crises in floating kidney present remote resemblances to acute appendicitis. 3. *Dysmenorrhœa.*—Menstrual colic, especially in an hysterical girl, may suggest appendicitis, but the seat of the pain, the period in the month, recurrence, the absence of a tumor, or the stigmata of hysteria are significant. 4. *Disease of the Right Tube and Ovary.*—A careful vaginal examination is necessary. The recognition of salpingitis or an enlarged, tender, prolapsed, or adherent ovary, together with a history of menstrual derangements or previous pelvic pain would be of diagnostic importance. The fact that such conditions are frequently associated with an infected appendix is by no means to be overlooked. 5. *Mucous colitis* is sometimes associated with chronic appendicitis without distinctive signs of the latter affection. In such a case I have seen a diseased appendix removed with decided improvement in the mucous disease and general health. 6. *Local abscess* in the cæcal region associated with malignant disease of the gut cannot always be distinguished from appendicular abscess. The anamnesis is important. An exploratory incision may be necessary. The situation of the tumor and œdema in perinephric abscess are of diagnostic importance. 7. *Intussusception, volvulus, and other forms of intestinal obstruction* present in the beginning only remote resemblances to appendicitis. Stercoraceous vomiting, so significant in these conditions, does not occur in appendicitis, nor do the tenesmus and bloody stools common in intussusception, especially in children, nor the sausage-like tumor, mostly on the left side, nor the invaginated gut upon rectal examination. 8. *Enteric Fever.*—There is no real difficulty in well-defined cases. Both the pain and tenderness are milder, vomiting is rare, fever is higher and the temperature tends to run a typical course, and headache is a prominent symptom. Later splenic tumor, rose spots, disproportion between the pulse-frequency and temperature, and the results of laboratory methods are

conclusive. The occasional occurrence of typhoid ulceration of the appendix and of appendicitis as an intercurrent affection in enteric fever is not to be overlooked. Perforation of the appendix may occur in the second or third week or during convalescence. 9. *General Peritonitis.*—In the absence of a history of the case the peritonitis which follows acute appendicitis cannot be differentiated from that due to other causes.

Prognosis.—The mortality is stated to be 2 per cent. of all cases, but the general statistics are without value since the death-rate is dependent upon extremely variable data relating to diagnosis and operation. The death-rate in early operations is low. The statistics vary from 2 to 10 per cent. according to the time of operation. The immediate recognition of the condition may be of vital importance to the patient. In cases characterized by recurrence, operation in a quiescent interval is attended with comparatively slight risk. The outlook in neglected cases is grave. The patient's general health may be undermined by prolonged suppuration and sepsis, or death may follow pylephlebitis or hemorrhage from an artery or vein. A very large proportion of cases in which early operation is neglected die of peritonitis. The fact that the course of the lesion in any given case can neither be foreseen from the beginning nor controlled by any other sure means, constitutes full justification for early operation.

viii. Enteroptosis.

Definition.—Falling forward or dragging downward of the intestines from stretching of their mesenteric attachments, practically always affecting the large intestine, and frequently associated with ptosis of the stomach, liver, and kidneys—*splanchnoptosis or Glénard's disease.*

Etiology.—See Gastroptosis.

Symptoms.—The general symptoms are often the only symptoms. The condition may exist for years without producing discomfort. The inherent weakness of the tissues which support the abdominal viscera is in turn aggravated by the gastro-enteroptosis. Persistent weakness, abdominal distress, pain in the back and loins, headache, torpor, dulness are common. The patients are always neurasthenic. Hyperchlorhydria or hypochlorhydria may occur. The local symptoms are varied. Abdominal discomfort, burning sensation, the weight of the prolapsed abdominal organs, colicky, irregularly recurring pains, nausea, anorexia, constipation, diarrhœa, abdominal distention are symptoms of enteroptosis. Obstruction from kinking is rare. Many of the so-called cases of intestinal indigestion belong here. Mucous diarrhœa and mucous stools are not uncommon.

Physical Signs.—These have been described under gastroptosis. In enteroptosis the transverse colon is the part of the intestine mostly affected and most readily demonstrated. Even on inspection the outline of the displaced bowel may be evident at and below the umbilicus. Peristalsis from right to left may be observed. Palpation will frequently reveal displacement of the liver and kidneys. At times the spasmodically contracted transverse colon can be clearly felt.

Diagnosis.—Direct.—The abnormal position of the colon, displacements of other organs, and diastasis recti are diagnostic features.

Inflation with the tube passed high up will often clearly distend the colon in its various positions. Simultaneous distention of the stomach prevents errors. The X-ray examination after barium injections may be of confirmatory value.

DIFFERENTIAL.—*Gastroptosis.*—These conditions commonly occur together. The absence of gastric symptoms and the normal position of the stomach on inflation may be observed in exceptional cases. *Malignant Disease of the Bowel.*—The long history of enteroptosis, ptosis of other organs, and diastasis recti, absence of marked anæmia or cachexia, absence of a tumor mass with palpable muscular contraction, absence of occult blood in the stools are in favor of enteroptosis.

Prognosis.—The condition may never cause trouble. A suitable abdominal support may relieve the symptoms but cannot permanently restore a prolapsed intestine to its normal position.

ix. Intestinal Indigestion.

Definition.—Disorders of digestion in the intestine, usually due to deficiency of the pancreatic ferments and characterized by the excretion of abnormal amounts of the various food matters ingested.

Etiology. — PREDISPOSING INFLUENCES. — Nervous disorders of the stomach or intestine predispose to intestinal indigestion.

EXCITING CAUSES.—Anacidity of the gastric juice, obstruction of the bile-ducts, pancreatic ducts, chronic intestinal disorders, chronic appendicitis, and dietetic errors are causes of intestinal indigestion. The definite cause that in many instances apparently interferes with the "secretin" production and the pancreatic supply is unknown.

Symptoms.—The general symptoms may be those of a neurosis: headache, coated tongue, persistent loss of weight without obvious cause or without symptoms. Habitual inability to digest certain articles is frequent. Pain in various regions of the abdomen, flatulence, and burning sensations are common. Constipation, diarrhœa, or pultaceous stools may occur. Foul-smelling flatus and fæces that decompose and ferment on standing are usual.

Physical Signs.—Abdominal distention may occur as the sign of active intestinal fermentation. Tenderness can usually be elicited. Examination of the fæces shows excess of fat and fatty acids, and much undigested proteid, or carbohydrate (see Fæces). The various tests (bead test, desmoid test, absorption tests) show impaired digestion and absorption.

Diagnosis. — DIRECT. — Chronic abdominal distress and symptoms of disturbance of nutrition, evacuation of abnormal amounts of undigested material as proved by chemical and microscopical tests, absence of physical signs of neoplasm, absence of blood from the stools and of fever are important diagnostic features.

DIFFERENTIAL. — *Intestinal Neuroses.* — The differentiation is often impossible as a neurosis may coexist with the intestinal indigestion. The condition of the fæces is important. Mucous stools must be distinguished from undigested material. *Neoplasm; Ulceration.*—Chronicity, absence of

blood from the fæces, absence of physical signs of obstruction or tumor, the persistent presence of undigested material in the fæces indicate intestinal indigestion.

Prognosis.—If the cause can be removed resumption of normal digestive power may return. The withholding of certain forms of food,—fat, carbohydrate or proteid, as indicated by the fæces,—may be continually necessary. Many cases persist unrelieved by treatment.

x. Intestinal Neuroses.

Definition.—Disorders of sensation and of the secretory and motor functions of the intestine.

Etiology.—Predisposing Influences.—All conditions which predispose to functional nervous disorders may be considered as predisposing influences.

Exciting Causes.—Shock or emotional outbreaks may precipitate an intestinal neurosis. The more common exciting causes are strain, worry, irregular habits of life, and dietetic errors.

Symptoms.—Symptoms common to all intestinal neuroses are nervous depression, exhaustion, and derangements of the normal function, sallowness of the complexion, and emaciation. When the digestive function of the intestines is not impaired a physical appearance of well-being is often seen. There is rarely any evidence in the fæces of impaired digestive activity. Common sensory symptoms are a feeling of weight or distention in the abdomen, the subjective perception of the intestine in peristalsis, persistent general discomfort or soreness in the intestines, and colicky pains which are often severe. These pains may simulate appendicitis, biliary, renal, or lead colic, or the tabetic crises. Motor disturbances give rise to nervous diarrhœa, intestinal rumblings and gurglings, or to spasmodic contractions of the bowel with constipation and pain. Explosive flatulence is a common motor neurosis. Retention of intestinal air or gases and distressing flatulent distention is frequently the result of intestinal spasm. Reverse peristalsis with fecal vomiting as a motor neurosis is rare. Few secretory neuroses are recognized. Diarrhœa and constipation are often due to motor causes. Membranous or mucous enteritis and colitis are practically the only secretory neuroses. The main features of these affections are severe abdominal pain, constipation, and the passage of large amounts of mucus, unformed and jelly-like, in opaque, firm flakes, or in large tubular casts. Bile-stained mucus is said to come from the small intestine; the paler variety from the large.

Several motor neuroses may occur in association. Physical examination usually yields negative results. In typical cases the abdomen may be scaphoid. Tenderness is common, particularly along the course of the colon. It is often marked over the cæcum. In mucous colitis, redness of the mucosa and excess of mucus can be seen on examination with the speculum.

Diagnosis.—Direct.—Persistent intestinal distress in a neurotic individual, the absence of the local signs of obstruction or neoplasm, the absence of anæmia and cachexia, a normal condition of the fæces as to

their constitutent fats, carbohydrates, and proteids, abnormal amounts of mucus, and the absence of parasites and their ova justify the suspicion of a neurotic basis for the intestinal disorder.

Differential.—*Intestinal Obstruction.*—A neurosis, even when associated with severe pain, constipation, and distention, rarely shows general symptoms, such as rapid pulse and collapse. Fever is absent, vomiting is less usual; local signs of obstruction are absent. The bowels are usually easily opened by the proper means. The detection of mucus in the stools in large amounts or in tubular form is important evidence in favor of a neurosis. *Intestinal New Growths.*—Tuberculosis of the intestine, anæmia, and cachexia are unusual in neuroses; local signs are absent. Tests for blood in the stools are negative. The general neurotic condition of the patient is a most important feature. In *gall-stone colic* subsequent jaundice, bile-stained urine, and detection of gall-stones in the fæces are important diagnostic points. Rapid pulse, fever, and persistent tenderness over the liver are usual in gall-stone attacks. In *renal colic* hæmaturia and occurrence of fever may be the only distinguishing features; in *lead colic* anæmia, "blue line," persistent constipation, and a history of exposure are important. With *tabetic crises* other features of tabes are present, for example, absent knee-jerks, Argyll-Robertson pupil, ataxia. *Appendicitis.*—Cases of intestinal neurosis are sometimes operated upon as appendicitis. The differentiation in some of the chronic cases may be extremely difficult. In the neuroses leucocytosis and muscular rigidity do not occur. Since mucous colitis is the condition most likely to simulate appendiceal disease mucous stools should be carefully looked for.

Prognosis.—The intestinal neuroses are refractory to treatment and often require years of careful management. Confirmed cases are often unrelieved by any form of general or local treatment.

xi. Intestinal Neoplasms.

New growths or tumors of the intestines, principally malignant. Benign tumors, lipomata, adenomata, myomata, polypoid growths also occur.

1. **Malignant Growths.—Carcinomata.**—Cancer of the intestines may involve any part of the bowel from the duodenum to the rectum. The large bowel is more frequently affected and especially the rectum. The growths are usually primary and tend to assume the annular form and to ulcerate, producing obstruction and hemorrhage.

Symptoms.—The general symptoms of malignant disease of the intestines are loss of appetite and strength, persistent and rapid loss of weight, early and severe anæmia, and rapidly developing cachexia. In many cases local symptoms are not recognized for a considerable time, a fact which lends importance to the general early derangements of health. The earlier local symptoms are those of partial obstruction and ulceration, colic, flatulent distention, and vomiting if the growth is in the duodenum, tenderness when it is situated in the large bowel. Malignant disease in the duodenum soon produces obstructive dilatation of the stomach with its characteristic symptoms. Persistent biliary vomiting indicates obstruction below the papilla of Vater. Duodenal cancer is often

associated with occlusion of the common bile and pancreatic ducts. Pain in the neighborhood of the growth itself, apart from the recurring colic of the obstruction-hypertrophy, is only marked in large-bowel cancers, and is of a dull aching or intense gnawing character. Hæmatemesis may occur in duodenal cancers. Black, tarry, or bright hemorrhagic stools are seen in the cases in which the growth is situated lower down. Diarrhœa, constipation, or dysenteric symptoms may be present in cancer of the lower bowel. Later local symptoms are those of complete obstruction, perforation, and metastatic deposits in the glands of the abdomen, in the liver, and elsewhere.

Physical Signs.—Careful and repeated inspection and palpation are necessary. Important signs are distention, particularly marked in low-lying tumors, intestinal patterns, visible peristalsis, and a visible or palpable tumor. Transmitted pulsation from the aorta may occur. Palpation with the flat of the hand, superficial at first, then deep, slowly covering the whole abdomen, is important in obscure cases. Small tumors may escape observation. Tumors of the duodenum may be central and do not move with the diaphragm; tumors of the jejunum and ileum are also usually central, but may appear in other regions, and are often freely movable. Tumors of the cæcum and transverse colon are movable but not so freely as those of the sigmoid flexure. Contraction of hypertrophied musculature, causing tension and hardening of the tumor mass, may be frequently observed. Movement of the tumor mass with respiration and with postural changes is, in the absence of adhesions, common to all tumors of the intestine save those situated in the duodenum and at the colonic flexures and rectum, and is readily appreciated by the examining hand. Palpable pulsation suggestive of an aneurism may be obtained over a tumor in relation with the abdominal aorta or the large iliac vessels. Tumor of the sigmoid may drop into the pelvis and be felt upon examination per rectum or per vaginam. During contraction of the intestine and forcing of the intestinal contents through the narrowing lumen, loud and sharp gurgling is often heard. Inflation of the stomach, slapping the abdomen with a cold wet towel will at times start up muscular action in the intestine and produce physical signs. Inflation of the sigmoid flexure and colon may bring a tumor into view, or the signs of stenosis of the bowel may appear. The X-ray examination frequently affords evidence of the presence and situation of a tumor. The characteristics of the vomitus in cases where the disease is situated in the duodenum are those of pyloric obstruction. The significance of biliary vomiting has been discussed. Occult blood is present when ulceration has occurred.

The fæces present no characteristic signs. Occult blood is always present with ulceration and is suggestive when local signs and symptoms are absent. Bloody mucous discharges and mucopurulent stools are frequent in tumors of the lower bowel. Carcinomatous tissue fragments are sometimes found in the stools when ulceration of the growth has taken place. The physical signs presented in examination by means of the proctoscope and sigmoidoscope are obstruction to the passage of the tubes, localized or annular thickening, thickened and ulcerated areas. Small tissue fragments may be removed and examined.

Diagnosis.—*Direct.*—Loss of appetite, persistent wasting, progressive anæmia are suggestive. Signs of obstruction, pain, colic, vomiting, distention, visible peristalsis, visible and palpable contractile tumor are indicative of a new growth in a muscular organ, as the intestine. Occult blood in the fæces or vomitus is an important sign of ulceration.

Differential.—Non-malignant Partial Obstructions.—The history of old inflammatory abdominal conditions may be suggestive of non-malignant obstruction. Absence of progressive anæmia and cachexia is an important point. The absence of hemorrhage, either occult or gross, particularly if the condition has persisted for some time, is in favor of a non-malignant condition. A tumor is rarely visible or palpable in non-malignant obstruction save just at the point of obstruction and at the time of contraction of the hypertrophied musculature. *Impacted Fæces, Scybala, etc.*—There is absence of anæmia and cachexia, wasting is unusual, signs of obstruction are commonly wanting. The evidences of hypertrophied intestinal musculature do not appear. Visible peristalsis is only present when obstruction is complete. Occult blood is not present nor do hemorrhages take place unless laceration of the mucosa of the rectum or anus has occurred. The scybalous tumor masses may be multiple and follow the course of the large bowel. They are not contractile but doughy and can at times be broken *in situ*. They can usually be removed by proper measures and are often readily distinguished by sigmoidoscopic examination.

Prognosis.—The outlook in intestinal carcinomata is hopeless unless the condition is early recognized and the case submitted to operation. In disease of the lower bowel, an artificial anus—colostomy—may prolong life for months; acute secondary accidents, as rupture of the bowel and complete obstruction, occur.

Sarcomata.—Sarcomata of the intestine are rare. They attain larger size, are less circumscribed and of more rapid growth than carcinomata. The general symptoms of anæmia and cachexia progress with alarming rapidity. Stenosis of the bowel does not occur, since the growth diffuses through large areas of the submucosa, hence visible peristalsis and contractile tumor masses are rarely observed. Hemorrhage is less common. The small intestine and the rectum are the favorite locations of intestinal sarcomata.

2. **Benign Tumors.**—The benign tumors, principally polypi, occur most frequently in the rectum and large bowel. Hemorrhage and tenesmus are the most marked features. The diagnosis of these and other benign tumors that lie out of reach of the sigmoidoscope must be attended with difficulty. Only when they reach large size do they produce signs of obstruction.

III. DISEASES OF THE LIVER.

i. Anatomical Anomalies of the Liver.

The contour of the liver is modified by the shape of the thorax and the pressure of adjacent organs. Abscess and tumors within the substance of the liver, as echinococcus, gummata, and malignant growths, cause departures from the normal form of the organ. In transposition of the viscera the shape of the liver is the reverse of normal. The convex anterior surface is frequently marked by parallel depressions corresponding to the ribs.

Remarkable changes in shape are produced by the permanent pressure of clothing—corset liver; lacing liver. That portion of the right lobe which is below the groove of compression may reach to the level of the crest of the ilium. It is separated from the main portion of the organ by a deep depression, the result of pressure atrophy, which produces in many cases great thinning, and in extreme cases an entire disappearance of the liver tissue, so that the corset lobe is connected with the liver by a flat band of connective tissue containing only bile-ducts and blood-vessels. The portion thus separated not only occupies an abnormal position, but it is also more or less freely movable according to the degree of atrophy of the compressed part. It, therefore, frequently simulates floating kidney, intestinal tumor, or other movable tumors occasionally found in this situation, especially when a loop of intestine has found its way into the groove. From these conditions it is to be differentiated by, (a) its ascent and descent with the respiratory movements of the diaphragm; (b) its continuity with the liver as demonstrable in many cases by percussion; (c) the continuous border of the anterior inferior surface of the liver and the corset lobe as recognized upon repeated, careful palpation, and finally, (d) by the presence of the notch or angle in the border at the point where the groove terminates anteriorly. Another deformity produced by the habitual pressure of clothing consists in an elongation of the entire right lobe downward. This change may be mistaken for enlargement of the organ. These changes are common in women but comparatively infrequent in men—belt liver.

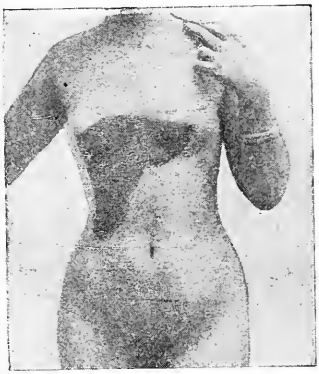


Fig. 316.—Corset liver.

Various changes in the position of the liver are encountered. These are usually the result of continuous pressure. Rotations upon the transverse axis may occur forward from the pressure of the clothing or backward from the pressure of abdominal tumors. The entire organ is frequently displaced downward, as in emphysema, pleural effusion, or subphrenic abscess, and upward by ascites, massive tympany, or ovarian or other abdominal tumors.

ii. Movable Liver.

Hepar Mobilis; Hepatoptosis.

Definition.—Marked displacement of the liver with abnormal mobility.

Etiology.—A slight degree of mobility occurs in enteroptosis and after large and long-continued ascites. True floating liver is extremely rare. It is associated with atrophy and relaxation of the abdominal walls and separation of the abdominal muscles, and large hernias in which the sac encloses many coils of intestines. The traction that the abdominal wall in pendulous abdomen exerts upon the liver by means of the ligamentum teres is a concomitant cause. Mechanical violence such as severe exertion or vomiting, persistent cough, falls, tight lacing, and rapid emacia-

tion have been regarded as etiological factors in floating liver. The frequency of these events as compared with the extreme infrequency of floating liver renders it in a high degree improbable that they play the part assigned to them in the causation of the latter condition. Therefore it is likely that floating liver can only occur in cases of congenital tendency to relaxation and elongation of the ligaments of the organ, or in which an actual mesohepar is present.

Symptoms.—There is, as a rule, no tenderness upon pressure. Pain is a common symptom. It is referred to the right hypochondrium and the epigastrium, and extends to the right shoulder and lumbar region. It is dull and dragging, and intensified by sudden movements. Spontaneous paroxysmal pain, bearing-down sensations, attacks of colic, with belching, meteorism, and constipation, and anomalous abdominal sensations are common. The pain is usually relieved by firm pressure upon the tumor or by lying down. Respiratory disturbances and palpitation occur. Hemorrhage from the stomach and bowels, ascites, hemorrhoids, and œdema of the legs and feet have been observed. Jaundice is rare, but the skin usually has a subicteroid hue.

Physical Signs.—The abdominal tumor occupies the right side and may extend as low as the pubic arch. The convex surface is directed forward and the entire organ is rotated to the right. The contour may be made out upon palpation. In the dorsal decubitus the liver may be replaced by gentle pressure unless fixed by adhesions—an extremely rare condition. Upon percussion when the liver is dislocated the pulmonary resonance passes directly into the tympany due to the intestines which have found their way into the space between the liver and the diaphragm.

Diagnosis. — A DIRECT DIAGNOSIS rests upon the position of the tumor, its contour, its large size, and the possibility of replacing the dislocated liver in its normal position. The diagnosis is greatly obscured by the presence of ascites and by the diminished mobility resulting from adhesions in the abnormal position.

The DIFFERENTIAL DIAGNOSIS involves the consideration of a greatly thickened mesentery and tumor of the kidney. Neither of these tumor masses is associated with tympanitic resonance in the normal area of liver dulness, nor can either of them be made by manual pressure to pass into the normal position of the liver. Floating liver occurs chiefly in women.

iii. Jaundice—Icterus.

This symptom-complex has been described in a previous section and the mechanism and significance of obstructive and toxæmic jaundice discussed.

The following special forms are of clinical interest:

1. ICTERUS PSYCHICUS (*Icterus ex Emotione*).—Sudden jaundice has been attributed to anger, fright, terror, and a gross insult. Associated symptoms are anxiety, epigastric distress, and diarrhœa. The jaundice passes away in a short time. The cases are not well authenticated and no wholly satisfactory explanation has been adduced for emotional jaundice.

2. HEREDITARY ICTERUS.—The cases are rare and may be regarded as clinical curiosities. Icterus neonatorum has been observed in every

member of large families in two generations. Another group includes cases of jaundice in a mother and three of her children, appearing in childhood and persisting for years without other evidences of ill health. The jaundice was of mild type. There was no enlargement of the liver or spleen.

3. Icterus Gravidarum.—Women in adanced pregnancy occasionally suffer from a form of catarrhal jaundice due to the pressure exerted upon the under surface of the liver by the enlarged uterus. Fecal accumulation acts as an additional cause of bile stasis, and the deformities of the liver resulting from lacing increase the tendency. The death of the fœtus or miscarriage may occur. The jaundice disappears after parturition.

4. Icterus Menstrualis.—Mild icterus has frequently been observed just prior to and during menstruation. When the discharge becomes free the jaundice abates. Enlargement of the liver and decolored fæces have been noted.

5. Biliousness. — Many persons, whose health is otherwise good, occasionally suffer from gastro-intestinal derangements, with headache, furred tongue, sensations of depression and malaise, and a subicteroid discoloration of the conjunctivæ or skin. Relief of these symptoms follows abstinence from food and mild purgation.

6. Starvation Jaundice. — Subicteroid discoloration of the conjunctivæ and skin is frequently present in persons who for any reason are unable to take food for several days. This is witnessed in insane persons who refuse food, and in stricture of the œsophagus, whether spasmodic or organic.

7. Syphilitic Jaundice.—Icterus appears in certain cases of severe syphilis coincidently with the secondary eruption. Its symptomatic character is shown by its prompt disappearance under antisyphilitic treatment. This form of jaundice is to be differentiated from accidental jaundice, such as occurs in tertiary syphilis as a consequence of diffuse hepatitis or gumma of the liver, and from a coincident catarrhal jaundice.

8. Icterus Following the Extravasation of Blood.—Yellowness of the conjunctivæ and skin may be observed after large hemorrhages into the skin or cavities of the body in scurvy, and after injuries or in lesions of the genital tract in women. The jaundice appears after several days, is not intense, and gradually fades in the course of some days or weeks. Bile pigments are present in the urine.

9. Icterus Following Hæmoglobinæmia.—This variety of jaundice is very marked after the attacks of hæmoglobinuria which follow exposure to cold or overexertion in persons suffering from malaria or syphilis. Fever, splenic enlargement, hæmoglobinuria, and jaundice constitute the symptom-complex. The urine contains bile pigments.

10. Toxic Icterus.—A large number of poisons are followed by icterus. Among them the following are important: arseniuretted hydrogen, certain mushrooms, toluylendiamin, glycerin, the bile acids, the chlorates, aniline and its derivative acetanilide, and the nitrites. Filix mas and santonin may cause a yellow discoloration of the skin. Icterus follows poisoning by phosphorus and lead. A very rapid icterus develops after snake bite.

11. Infectious Icterus. — Yellow fever and relapsing fever are characterized by marked icterus. Septic conditions and pneumonia are frequently, enteric fever occasionally, attended by jaundice.

12. Epidemic Icterus.—When a number of persons living under the same conditions develop jaundice, as occasionally occurs in boarding schools, camps, barracks, or prisons, the term epidemic jaundice is warranted. Many large and small epidemics have been described in the literature. These epidemics are usually of short duration; in a few instances they have lasted several months. The disease commonly assumes the guise of ordinary catarrhal jaundice and runs a benign course; in some instances it is severe and many deaths occur. In pregnant and parturient women the prognosis is grave. Epidemic icterus has been attributed to atmospheric or climatic influences, dietetic faults, and infectious causes. A combination of these agents may be operative. (See Icterus Infectiosus.)

13. Postvaccinal Jaundice. — In rare instances jaundice has appeared in groups of cases among revaccinated persons. The jaundice has occurred at intervals of a few days to several months. It has been attributed to wound infection. This form of epidemic jaundice is rare and its association with vaccination is probably accidental. It is much more likely due to other local influences affecting groups of persons who happen to have been vaccinated.

iv. Icterus Neonatorum—Physiological Icterus.

Definition.—A variety of icterus common in the new-born occurring independently of any particular disease or lesion and pursuing a favorable course.

Etiology.—This affection occurs in about one-half of all babies. It is more common in foundling hospitals than in private practice, in premature infants than in those born at term, in boys than girls, and in cases where parturition has occurred under chloroform. The pathogenesis of the condition is not clear. It has been attributed to rapid destruction of erythrocytes after birth, to stasis in the smaller bile-ducts, to resorption of bile from the intestine, and to œdema of the periportal connective tissue.

Symptoms.—The jaundice appears upon the second or third day after birth and first upon the face and chest, rapidly extending to the rest of the body. It fades more or less rapidly in the course of ten or twelve days. The general condition of the child is otherwise normal. The urine is normal and does not contain bile pigment in solution. The stools, after the discharge of meconium, have their usual golden-yellow color. The pulse-frequency is not lowered.

Diagnosis. — Direct. — The comparative mildness of the jaundice and the complete absence of serious symptoms suffice to establish the nature of the affection. Its gradual disappearance within two or exceptionally as late as three or four weeks is not followed by recurrence.

Differential.—In the following forms of jaundice in the new-born the discoloration is more intense and associated with serious symptoms: (a) congenital absence of the common or hepatic duct; (b) congenital syphilitic hepatitis, in which the characteristic external lesions of syphilis

are also manifest, and (c) septic infection by way of the umbilical vein, a fatal form of sepsis associated with phlebitis and, in some instances, with umbilical hemorrhage. Icterus may occur in the new-born as a result of obstruction of the bile-ducts, acute fatty degeneration of the liver, and epidemic hæmoglobinuria.

Prognosis.—The physiological icterus of the new-born is never fatal.

v. Acute Yellow Atrophy.

Malignant Jaundice; Icterus Gravis.

Definition.—An acute disease characterized anatomically by diffuse necrosis of the liver-cells with great diminution in the size of the organ, and clinically by intense jaundice and cerebral symptoms.

Etiology. — Predisposing Influences. — Acute yellow atrophy is rare. It is more common in women than in men in the proportion of about 8 to 5. This difference is in part explained by the fact that pregnant women are sometimes affected after the fourth month or at the time of parturition. The greater number of cases occur between the twentieth and fortieth years. Acute yellow atrophy is comparatively rare among children. It has followed osteomyelitis, erysipelas, sepsis, enteric and relapsing fever, and the early stages of syphilis. It has been attributed to ptomaine and mushroom poisoning, to alcohol and to chloroform, to fright, and to profound depressing emotions. The symptoms caused by phosphorus poisoning resemble those of acute yellow atrophy, but the conditions are neither etiologically nor pathologically identical. In rare instances acute yellow atrophy has occurred as an intercurrent disease in hypertrophic cirrhosis, bile stasis, and fatty degeneration of the liver.

The Exciting Cause.—The actual pathogenic principle is unknown. Various micro-organisms, especially streptococcus and Bacillus coli, have been found in the liver, but in many of the cases examined no bacteria have been present.

Morbid Anatomy.—The liver is greatly reduced in size, flaccid, and folded upon itself. Its capsule is wrinkled and of a dirty yellowish-green color. Upon section the surface is mottled and the outlines of the lobules are indistinct. These are yellowish masses surrounded by a dark reddish tissue, the latter representing a more advanced stage. Microscopically the hepatic cells are indistinct, bile stained, and in all stages of granular and fatty degeneration and necrosis. The capillary vessels and bile-ducts are destroyed, with resulting minute hemorrhages and extravasation of bile.

Symptoms.—The attack usually begins as an acute gastric catarrh, which is shortly followed by more or less intense jaundice with clay-colored stools—the initial stage. In the course of some days or, less frequently, two or three weeks, during which the symptoms have remained comparatively mild, the second stage sets in suddenly with vomiting, restlessness, stupor, delirium, convulsions, and coma. Hemorrhages into the skin and from mucous surfaces are common. The jaundice becomes more intense. Pregnant women usually abort. There may be pain in the region of the liver. The temperature is normal or subnormal, rising toward the

end. Exceptionally there is marked fever throughout the attack. The tongue is coated and dry. The action of the heart, normal or slow in the initial stage, becomes rapid and feeble in the second stage, with enfeeblement of the first sound and not rarely a soft, blowing, systolic murmur. The liver, which is enlarged in the first stage, undergoes, with the development of the cerebral symptoms, a rapid diminution in volume. There may be complete absence of hepatic dulness, as the flaccid organ folds upon itself and falls away from the abdominal wall, coils of intestines taking its place. The splenic dulness is increased. The abdomen is very sensitive, particularly in the epigastric zone, and there is spontaneous pain. The urine is slightly decreased, contains bile pigments, generally small quantities of albumin and tube-casts. Products of disordered metabolism, such as leucin, tyrosin, and sarcolactic acid, are also present. Urea is much diminished and the percentage of nitrogen present as ammonia correspondingly increased. Leucin and tyrosin are sometimes absent from the urine. Albumoses are sometimes present in small amounts.

Diagnosis.—Direct.—Intense jaundice, vomiting, diminished area of liver dulness, hemorrhages, enlarged spleen, grave cerebral symptoms, together with leucin and tyrosin in the urine, constitute a characteristic symptom-complex. The initial stage cannot be distinguished from ordinary gastroduodenal catarrh with jaundice.

Differential. — *Hypertrophic Cirrhosis.* — In rare cases there are intense cerebral symptoms, but enlargement of the liver, fever, the absence of leucin and tyrosin, and the long course of the disease prior to acute cerebral symptoms are distinctive.

Acute Phosphorus Poisoning.—The symptoms may be almost identical, particularly in respect of hemorrhages, jaundice, and decrease in liver dulness, but the gastric symptoms are usually more intense and set in directly after the ingestion of the poison without a prodromal or initial stage, the icterus rather abruptly on the third day, and leucin and tyrosin are said to be absent from the urine. A dilated transverse colon may so displace the liver upward as to simulate atrophy, but the gradual reduction in the area of liver dulness from day to day is most significant.

Prognosis.—The disease is almost always fatal. The outlook is more unfavorable when cerebral symptoms occur early. The duration of the illness varies from a few days to two or three months. About half the cases die between the fifth and fourteenth days, about one-third within five weeks. The duration of the characteristic stage varies from one or two days to a week. In the cases that run a favorable course the cerebral symptoms are less violent and the duration of the disease is prolonged.

vi. Diseases of the Bile Passages and Gall-bladder.

CATARRHAL JAUNDICE.

Icterus Gastroduodenalis; Icterus Simplex.

Definition.—Jaundice due to swelling and mucus in the intestinal portion of the common duct, the result of the extension of gastro-intestinal catarrh.

Etiology.—Predisposing Influences.—Catarrhal jaundice is a common affection and is probably always associated with catarrhal inflammation of the duodenal mucosa. All conditions which predispose to the latter affection therefore favor its occurrence. Chief among these are chronic alcoholism, chronic gastric catarrh, conditions favoring portal obstruction, chronic valvular disease, and chronic nephritis. Malaria is a well-recognized cause. The affection also frequently occurs in connection with the acute infections, particularly enteric fever and pneumonia.

Age exerts an important predisposing influence. Catarrhal jaundice is a disease of young persons and is rare in middle and advanced age. It is common in children after the second year. In adult life it is more common in males than females.

The Exciting Cause.—The immediate cause is usually an acute indigestion, sudden cold or exposure, or unusual physical strain with irregular meals. In many cases no causal factor can be discovered. When a number of persons are exposed to the same local influences, as in a school, a circumscribed epidemic of catarrhal jaundice may occur.

Morbid Anatomy.—The mucous membrane of the terminal portion of the common duct is swollen and the ampulla of Vater may be obstructed by a plug of tenacious mucus. It is possible that the catarrhal process may invade the smaller ducts, but of this we have no definite knowledge.

Eppinger recently found in the case of a girl who was instantly killed by an accident on the ninth day of an attack of catarrhal jaundice that the portion of the common duct which lies within the wall of the intestine was impermeable and that the occlusion was due to hyperplasia of the lymphoid tissue of the mucosa of the duct. It is probable that certain cases of catarrhal jaundice are due to inflammatory swelling; others to the presence of a plug of tenacious mucus, and yet others to a hyperplastic condition of the lymphoid tissue which surrounds the mucous glands of the appendix in varying proportion in different individuals.

Symptoms.—In many cases the jaundice is preceded by epigastric distress, loss of appetite, a coated tongue, and nausea and vomiting. Not rarely there are also present headache, vertigo, mental depression. Fever is not common, but the temperature may reach 101°–102° F. (38.3°–38.9° C.). The bowels are constipated and the stools light in color but rarely entirely free from bile pigment. The urine contains bile pigments and is scanty and sedimentary but not often albuminous. Hyaline casts are common. The skin and conjunctivæ are more or less deeply jaundiced, but the olive green of chronic jaundice does not occur. The nervous symptoms of jaundice are present in varying degree, especially pruritus and drowsiness. Slowing of the pulse and respiration is less frequent. The liver is usually slightly enlarged. The gall-bladder is rarely palpable. Slight enlargement of the spleen is not uncommon. There are cases in which the above-symptoms do not occur and the patient's knowledge of his being jaundiced is obtained from the looking-glass or the inquiries of his friends. The duration of the affection is from two to six or eight weeks, the jaundice gradually fading, the bile pigment first reappearing in the stools, next disappearing from the urine and finally from the sclera. There are cases of catarrhal jaundice which last two or three

months with remissions and exacerbations, but the diagnosis in such cases must be guarded, especially in elderly persons.

Diagnosis.—Direct.—The diagnosis may usually be made without reserve from the youth of the patient, his previous fair health, the symptoms of gastric catarrh, the moderate intensity of the jaundice, and the short duration of the affection.

Differential.—The diagnosis from cirrhosis, cholelithiasis, carcinoma, and Weil's disease is considered under those respective headings, to which the reader is referred. It is important to remember that catarrhal jaundice is rare in old persons and that a jaundice persisting beyond six or eight weeks can only be regarded as catarrhal after a rigid process of exclusion in regard to all other possible causes.

Prognosis.—The outlook in simple uncomplicated catarrhal jaundice is highly favorable. It is a benign affection.

CHRONIC ANGIOCHOLITIS.

Definition. — Chronic inflammation of the bile-ducts. It may be catarrhal or suppurative.

1. **Chronic catarrhal cholangitis** may occur as a sequel of acute catarrh. It is always combined with obstruction of the common duct, and is therefore an associated condition in cholelithiasis, parasites, cancer, stricture, and compression of the common duct from without. The obstruction may be complete or incomplete.

(a) *Complete Obstruction.*—The bile passages, the gall-bladder, and the intrahepatic ducts are dilated and contain clear mucus, which is usually sterile. The patients are persistently and deeply jaundiced. Fever is commonly absent.

(b) *Incomplete Obstruction.*—There are one or more calculi in the common duct and, as a rule, in the gall-bladder, or there is pressure from the outside. The bile may escape in small amounts continuously, or the obstruction may be intermittent. The bile passages and gall-bladder are not usually greatly dilated. They contain a thin, bile-stained mucus. The jaundice may vary in intensity and the stools show the presence of bile pigments. Febrile attacks,—hepatic fever,—characterized by chills, rapid rise of temperature, and profuse sweating, are common in this form of obstruction and are caused by infection.

2. **Suppurative cholangitis** affects the large and small ducts. In the majority of the cases the gall-bladder is also involved. There is dilatation of the bile passages and particularly of the common ducts. The walls are thickened. The intrahepatic ducts are much dilated, and minute collections of pus mixed with bile are formed by the suppurating ducts and disintegrating hepatic tissue. There is usually distention of the gall-bladder, which is filled with pus, occlusion of the cystic duct, and adhesive inflammation of the gall-bladder to adjacent parts. Suppurative cholangitis constitutes one of the most serious complications of cholelithiasis. It occurs also in consequence of the presence of foreign bodies, as fish bones, or intestinal parasites, as ascarides, which find their way into the ducts from the intestine, and in connection with cancer of the ducts. It is a somewhat rare sequel of enteric fever, pyæmia, and dysentery.

The onset is insidious. The symptoms vary in intensity and are not always characteristic. As a rule they are severe, consisting of jaundice, enlargement of the liver, pain and tenderness in the region of the gall-bladder, which is often distended, fever of septic type, and a marked leucocytosis. There is commonly a history of biliary colic. Pylephlebitis, endocarditis, purulent meningitis, and peritonitis are occasional complications.

The diagnosis of suppurative cholangitis rests upon a history of gall-stones, jaundice, intermittent fever of hectic type, tenderness and pain in the region of the gall-bladder. The fever is to be distinguished from malaria by the leucocytosis, the variable periodicity, and above all by the absence of blood parasites. A tender point in the region of the twelfth dorsal vertebra, 2.5 to 3 cm. from the middle line, may be present in inflammation of the bile-ducts (Boas). In cases occurring in association with the acute infections jaundice may be absent or slight. The differential diagnosis between certain cases of suppurative cholangitis and abscess of the liver may be attended with difficulty. In favor of the latter condition are absence of jaundice, slight fever or even subnormal temperature, and the absence of tenderness and pain in the region of the gall-bladder.

The outlook is highly unfavorable. The reëstablishment of biliary drainage by the escape of the stone or its removal by operation may be followed by recovery. In a case recently under my observation operation failed and there were many small calculi pocketed in abscess cavities throughout the liver.

VARIOUS LESIONS OF THE BILE PASSAGES.

It is convenient to consider ulceration, perforation, stricture, and fistulæ of the bile-ducts in connection with cholelithiasis, which is their usual cause.

The lumen of the common duct may be partially or completely occluded by the seeds of fruit and by certain parasites, among which lumbricoid worms are common and echinococci and distomata rare causes of obstruction in man.

Obstruction by pressure from without is more common. Carcinoma or fibroid thickening of the head of the pancreas, or, in rare instances, cancer of the pylorus, enlarged lymph-glands, abdominal tumors, and aneurism of the cœliac axis may compress the common duct.

The symptoms have already been described in connection with complete and incomplete obstruction under the heading Chronic Angiocholitis. Complete permanent occlusion of the common duct terminates in death. The conditions which cause occlusion by pressure from without are usually fatal. The diagnosis of the cause of the obstruction may be difficult. Colic, with variable jaundice and intermittent fever, suggests cholelithiasis. Cancerous disease in the rectum or genito-urinary tract, or the stomach or intestines, points to secondary glandular infiltration as the cause of biliary stasis and jaundice. Accessible groups of lymph-nodes and, in particular, the retroclavicular lymphatic glands may also be enlarged. The gall-bladder is frequently distended and may be distinctly palpable.

INFLAMMATION OF THE GALL-BLADDER; CHOLECYSTITIS.

The condition may be acute or chronic.

1. **Acute Cholecystitis.**—The inflammation may be catarrhal, suppurative, or phlegmonous. These forms sometimes represent different degrees of intensity.

Etiology. — Acute cholecystitis is commonly due to gall-stones. It may, however, result from bacterial invasion in the absence of cholelithiasis. It is common in the infectious fevers and a subacute form is very often met with in enteric fever. The usual pathogenic organisms are the colon bacillus, the bacillus of Eberth, the pneumococcus, streptococcus, and staphylococcus. The condition is frequently associated with cholangitis and dilatation of the bile passages. The gall-bladder is usually distended. In subacute cases of long duration distention may be prevented by fibrous thickening of the walls. Adhesions with the adjacent parts of the liver or the omentum or colon may take place. The cystic duct is frequently occluded even in the absence of an impacted calculus. The enlargement sometimes takes place upward and inward and there is no palpable tumor. The contents may be a thin, dark-greenish mucus, or mucopurulent, purulent, or hemorrhagic. Perforation may take place, with abscess formation limited by the adhesions or into the peritoneal cavity.

Symptoms.—In the milder forms there may be simply some tumefaction with dulness, circumscribed tenderness, and a rise of temperature. This form is common in enteric fever. The severe forms are ushered in with intense paroxysmal pain in the region of the gall-bladder, the epigastrium, or in the right upper quadrant of the abdomen. With this are associated nausea and vomiting, arrest of peristalsis, rigidity of the abdominal muscles and especially of the rectus upon the right side, and fever. The enlarged gall-bladder may be sometimes recognized upon palpation and percussion, but as a rule the extreme tenderness interferes with physical examination. In the absence of gall-stones jaundice does not commonly occur.

Diagnosis. — DIRECT. — The milder forms are readily recognized; in the more severe cases the condition is often very obscure. The anamnesis is important. The above symptom-complex occurring in a patient who has had attacks of hepatic colic or cholecystitis, or who is convalescent from enteric fever or pneumonia, is significant. The recurrent forms are readily diagnosticated. It is important to remember that cholecystitis may occur without gall-stone disease.

DIFFERENTIAL.—The condition may simulate acute obstruction of the bowels or appendicitis. While these conditions may be differentiated from acute cholecystitis by characteristic symptoms in a large proportion of the cases, there are instances in which the actual organ affected has been revealed only upon operation.

Prognosis.—The outlook depends upon the intensity of the inflammatory process. The purulent and phlegmonous forms are usually fatal Timely surgical intervention may be the means of saving life. The danger of perforation is to be constantly borne in mind.

2. **Chronic Cholecystitis.**—The common cause of chronic cholecystitis is cholelithiasis. The disease may arise in consequence of extension of the inflammation in cholangitis. The muscular and connective-tissue elements of the wall are involved. When the contents undergo resorption, or escape through the cystic duct or by way of a fistulous opening, the thickened wall contracts and the gall-bladder becomes permanently reduced in size. Its walls under these circumstances are sometimes the seat of calcareous changes. Pericholecystitis may develop with diffuse swelling around the organ, and later fluctuation. The cystic duct may be completely occluded by a calculus or by cicatrices. The bile is then absorbed, the mucous membrane, however, continues to secrete an abnormal mucus, and the gall-bladder undergoes gradual distention with thickening of its walls and sometimes more or less extensive peritoneal adhesions. The contained liquid may be light in color and bile-free,—dropsy of the gall-bladder,—or it may be pus—empyema. Gall-stones are frequently present.

Symptoms.—When the dilatation is slight the gall-bladder extends below the border of the liver but cannot be palpated unless the abdominal walls are thin. As the enlargement proceeds it constitutes a palpable pear-shaped tumor, which is movable from side to side and may be displaced backward by moderate pressure, but which resumes its position when the pressure is withdrawn. The enlarged gall-bladder moves upward and downward with the respiratory play of the diaphragm and partakes of the movements of the liver. It may be greatly enlarged and elongated, and instances have been noted in which the contents have amounted to a litre. When the fundus presents toward the abdominal wall and a loop of intestine has found its way into the space between the fundus and the liver, the condition may simulate an echinococcus or ovarian cyst or hydronephrosis. If the walls of the abdomen are thin and relaxed, the tumor formed by a dilated gall-bladder may be visible. Urgent as the symptoms attending the disease which has caused the dilatation may have been, the condition itself usually causes no important subjective symptoms. When pain and tenderness are present they are commonly due to local adhesions and peritonitis.

Diagnosis.—Direct.—The diagnosis may be difficult. The anamnesis, the palpable and visible tumor connected with the liver and partaking of its movements, its cystic nature, its elasticity, its gourd-shaped outline, its mobility and tendency to at once resume the position from which it has been manipulated constitute adequate data for a positive diagnosis. The nature of the contents can only be ascertained by their removal. For this purpose an exploratory cœliotomy can be performed,—never an exploratory puncture, which is attended with the danger of the escape of a portion of the fluid into the peritoneal sac.

Differential.—The diagnosis from an echinococcus cyst may be attended with great difficulty. The hemispherical form, more restricted movements, and hydatid thrill are significant. In hydronephrosis the deeper origin, relatively slighter mobility, except in floating kidney, and the outline are of diagnostic importance, and the occasional disappearance of the tumor with great diuresis seen in intermittent hydronephrosis would be distinctive. Ovarian cysts spring from the pelvis and can be shown

by vaginal and bimanual examination not to be connected with the liver but with the uterus.

Prognosis.—In many cases it is favorable after the tumor has ceased to enlarge and is of moderate size. The inconvenience resulting from its presence and pressure upon adjacent organs, and the danger of adhesions and fistula formation, and, in particular, the danger of rupture justify drainage or excision, which is often followed by complete restoration to health.

CANCER OF THE BILE-DUCTS AND GALL-BLADDER.

Primary malignant disease of the gall-bladder is commonly associated with gall-stones—70 to 100 per cent. according to various statistics. The symptoms of the condition often gradually supervene upon those caused by biliary calculus. The fundus is often the starting-point of the growth, which early becomes manifest as a dense tumor in the region of the gall-bladder, developing downward and toward the median line, and not movable by reason of firm adhesions and implication of the surrounding tissue. The mass may attain large dimensions. The gall-bladder is sometimes greatly distended. The condition is much more common in females than males and rarely appears before the fortieth year of life. Pain is a prominent symptom. It is often paroxysmal but continues throughout the intervals. There is also more or less tenderness. Jaundice is present in a majority of the cases. It may be due to implication of the ducts or to pressure upon their walls.

Primary cancer of the bile passages is comparatively rare. The common duct is usually the starting-point of the growth, which may involve the walls of the ampulla of Vater and invade the hepatic and cystic ducts. There is rarely a palpable tumor. Jaundice occurs early and is persistent. If the carcinomatous infiltration involves the portal vein ascites results. There is often profound anæmia, but early cachexia may not be present. Cholæmia is a common terminal condition. Extension to the liver gives rise to symptoms characteristic of carcinoma of that organ.

CHOLELITHIASIS.

Gall-stone Disease.

Definition.—A condition characterized by the formation and presence of biliary calculi in the gall-bladder or bile passages. The great majority of gall-stones are formed in the gall-bladder.

Etiology. — Predisposing Influences. — All conditions which give rise to the stasis of bile in the gall-bladder predispose to cholelithiasis. The outflow of bile may be impeded by partial or complete occlusion of the bile-ducts by catarrhal swelling of their mucous membrane, the presence of calculi or parasites, adhesions in the region of the porta, or compression by enlarged lymph-glands, the head of the pancreas, or the duodenum. Atrophy of the musculature of the gall-bladder from distention or age may lead to stagnation of the bile. Lacing plays an important part, (a) by restricting the movements of the diaphragm, (b) by causing elongation of the liver, displacement of the gall-bladder, and bending or twisting of

the cystic duct, (c) by inducing changes in the anatomical relations which expose the cystic duct to compression, especially when there is, as is frequently the case, displacement of the right kidney, and (d) by causing gastroduodenal catarrh which may be followed by cholangitis and cholecystitis. Relaxation of the abdominal walls and enteroptosis favor the stagnation of bile in the gall-bladder. Lack of exercise, prolonged rest in bed in convalescence from acute or in chronic disease, and sedentary occupations constitute predisposing factors of importance, especially when combined with overfeeding and constipation. Cardiac affections, and in particular mitral stenosis, predispose to gall-stone disease by the passive visceral congestion and catarrhal processes to which they give rise and by the sedentary life which they enforce. There are great differences in the prevalence of gall-stone disease in different localities and different countries, as determined by post-mortem statistics,—a fact ascribed to local differences in mode of life, occupation, and the influence of endemic diseases, which by causing gastro-intestinal catarrh may become indirect factors in the production of calculi. The rare cases reported in the newborn and in infancy are simply clinical curiosities. The disease is rare under thirty. The liability increases progressively after forty. Women suffer more frequently than men in the proportion of 5 to 2. The pressure of the pregnant uterus upon the bile-ducts and its interference with the movements of the diaphragm, and the relaxation of the abdominal wall after frequent pregnancies are to be considered. The more sedentary life of women constitutes a predisposing influence of importance.

The Origin of Gall-stones.—The theory of Naunyn is generally accepted. A catarrhal condition of the mucosa of the gall-bladder, leading to an increased formation of cholesterin and lime salts, is the primary cause of the formation of gall-stones. This lithogenous catarrh may be produced by various causes, but the most important factor in its production is the presence of various bacteria. Among those which have been isolated are the colon bacilli, streptococci, staphylococci, pneumococci, and typhoid bacilli. They may gain access to the gall-bladder by way of the blood, or from the intestine by way of the common and cystic ducts. They have been demonstrated in the centre of gall-stones. Cholesterin, lime salts, and bilirubin deposited around collections of epithelial débris and bacteria constitute the beginnings of biliary calculi. The masses thus formed grow in size by the gradual accretion of similar substances. Gall-stones have been experimentally produced by the injection of cultures of bacteria into the gall-bladder of animals. The above facts account for the frequent occurrence of cholelithiasis after the acute infectious fevers, especially enteric fever.

The Chemical and Physical Characters of Gall-stones.—Gall-stones are composed chiefly of cholesterin, bilirubin in combination with calcium, and calcium carbonate. These constituents are present in varying proportions. Ordinary calculi consist of 70 to 90 per cent. of amorphous or crystalline cholesterin. The small, dark stones found in the ducts are principally composed of pigment in combination with calcium and calcium carbonate. Free bile pigment is not usually present. Traces of iron, manganese, and copper, and bile acids and fatty acids are also present. The

color of the stones is not uniform, but irregular, and depends upon the quantity, character, and mode of deposit of the pigment which they contain. White or pale fawn-colored calculi consist of nearly pure cholesterin. Other stones may be yellow, greenish, or brown. An excess of pigment may give them a dark reddish-brown or black color. The cortex, main body, and nucleus are usually colored differently. The consistency also varies. Cholesterin stones are often so soft that they may be crushed between the fingers. Recently formed concretions are commonly soft; older ones may be harder with a soft central nucleus. The outer layer may be hard and enclose an unformed mass of cholesterin. The larger the proportion of lime salts the harder the calculus. Section usually shows the cholesterin to be deposited in concentric layers with radiating crystalline striæ, the result of recrystallization.

Gall-stones vary in number from one to hundreds or even a thousand or more. Single stones are usually ovoid and may be of large size—3 or 4 cm. or, in one reported instance, 7.5 cm. in long diameter. The solitary stone is usually closely embraced by the gall-bladder. Multiple calculi are commonly polygonal, with smooth, faceted surfaces, and owe this form to the pressure exerted among themselves while soft. Traces of faceting may be seen in the small calculi numbered by hundreds occasionally met with. It sometimes happens that a small number of ovoid, unfaceted calculi are found. They are of the dense variety and consist largely of the bilirubin-calcium combination. When this form is present in great numbers, the individual stones are not larger than small shot and are spoken of as gall-sand. Gall-stones impacted in the ducts sometimes undergo enlargement by further accretions of cholesterin and lime salts. Stones are found in this situation of such a size that they could not have passed through the cystic duct. Small, ovoid, greenish-black calculi are sometimes found in the intrahepatic bile-ducts, especially in cirrhosis of the liver. In a majority of the instances they are all of the same variety and composed of bilirubin-calcium. In fact, in cases in which numerous gall-stones are present, they are almost always all of the same variety.

Symptoms.—The subject of cholelithiasis may be clinically considered under the following headings: gall-stones quiescent in the gall-bladder; the symptoms which attend the passage of a stone through the ducts; the symptoms produced by the permanent obstruction of the ducts; ulcerative lesions caused by gall-stones; and gall-stones in the intestines.

1. **Gall-stones Quiescent in the Gall-bladder.**—In a great majority of cases biliary calculi, so long as they remain in the gall-bladder in which they are formed, produce no symptoms. Their presence in this viscus is frequently discovered at the autopsy in cases in which they have caused no manifestations whatever during life. According to Kehr symptoms occur in only about 5 per cent. of all cases. Persons who suffer from repeated attacks of biliary colic frequently have no trouble from them during the intervals. In a small proportion of the cases there are symptoms which suggest their presence, even though they are not sufficiently characteristic to justify a positive diagnosis. When, however, such symptoms occur during the intervals between attacks of colic, especially when such attacks have been followed by the passage of faceted calculi, their signif-

icance is clear. These symptoms consist of subjective sensations of weight in the right hypochondrium, aggravated some hours after taking food, frequent dull pain in the region of the gall-bladder radiating toward the right shoulder and the lumbar region, and nervous and mental derangements such as are common in neurasthenia—depression, irritability, precordial and epigastric distress, and headache, coryza, and flying neuralgic pains. Upon physical examination the gall-bladder may sometimes be found to be enlarged and palpable, and in very rare instances a crepitus caused by the movement of multiple calculi among themselves may be detected.

Gall-stones in the intrahepatic ducts rarely give rise to symptoms. If numerous or large they may occasion pain, enlargement of the liver, or jaundice, but these symptoms are not of diagnostic value. When infection takes place they cause diffuse intrahepatic cholangitis.

2. **The Symptoms Attending the Passage of a Gall-stone Through the Ducts.**—Gall-stones occasionally become arrested in the cystic or the common duct without causing pain. Small stones may traverse these passages without giving rise to colic. This has been observed in cases in which the repeated passage of larger stones is inferred to have caused a gradual dilatation of the ducts. When stones of a larger size are passed by the bowel in the absence of a history of colic, it is probable that they have reached the intestine by way of a fistulous communication with the gall-bladder or ducts.

Biliary Colic.—Commonly the passage of a gall-stone is attended by the symptoms of gall-stone colic. The attack usually begins with violent pain in the right hypochondrium with its focus of intensity in the region of the gall-bladder. In some cases the pain is referred to the epigastrium or the lower thoracic region, or on both sides, or to the right mammary region. It may radiate toward the abdomen or back, and occasionally to the right shoulder. It is usually agonizing and the patient groans and rolls about in uncontrollable distress, or he may twist his body to the right, or sit with his thighs and knees strongly flexed and his body bent forward so as to relax the abdominal muscles. There may be temporary remissions of pain which are followed by exacerbations of greater violence. The gall-bladder is often palpable and tender, and the liver may be somewhat enlarged, with tenderness over the hepatic area. Vomiting, chills or chilliness, a rise of temperature sometimes to 103°–104° F. (39.5°–40° C.), profuse sweating, and great general relaxation occur. In cases marked by high fever there may be enlargement of the spleen and febrile albuminuria. It is probable that there are under these circumstances bacterial invasion and acute cholecystitis. The fact that the symptoms of gall-stone colic are sometimes present in acute cholecystitis without gall-stones is not to be overlooked. Jaundice is a common symptom. It does not occur so long as the stone is engaged in the cystic duct, but follows the lodgement of the stone in the common duct. When the stone is of small size and passes rapidly through the common duct into the intestine, jaundice may not occur. In any case jaundice does not occur until several hours, often twenty-four, have elapsed from the beginning of the attack. It is usually transient, but may persist for several days or weeks. The jaundice is the very type of

obstructive jaundice and is associated with clay-colored stools, the presence of bile pigments in the urine, itching of the skin, and other characteristic symptoms.

The duration of the attack varies from a few hours to several days. When the stone escapes into the intestine the pain ceases, often as abruptly as it began, leaving some degree of local tenderness, which rapidly subsides, and lassitude, from which the patient gradually recovers. Not rarely the stone lodges in the ampulla of Vater and acts as a ball-valve, causing recurrent attacks of pain and jaundice. The pain is a true colic caused by the spasmodic contraction of the musculature of the bile-ducts and the violent pressure of the stone upon the mucous membrane. The swelling and tenderness of the gall-bladder and liver are due to bile stasis and consequent distention of these organs. In bacillary invasion there is the superadded pain of inflammation. Rare accidents are fatal syncope and the rupture of the gall-bladder into the peritoneal sac. Palpitation and precordial distress may occur, while general convulsions and hysterical seizures are occasionally observed in neurotic subjects.

Direct Diagnosis of Biliary Colic. — The diagnosis rests upon the location of the focus of pain, its radiation, local tenderness, the abrupt onset of the attack, vomiting, chill or chilliness, with fever and the symptoms of obstructive jaundice. The history of previous attacks is suggestive; the presence of gall-stones in the stools is conclusive. Their absence is, however, only of negative importance in diagnosis. It may be due to a faulty method of examination, to the return into the gall-bladder of a stone which has engaged in the cystic duct, to cessation of muscular spasm in the walls of the ducts, to the passage of the stone from the narrow cystic duct into the wide common duct and its retention there, and, finally, to the disintegration of the stone in the intestine.

The stools must be thoroughly stirred with a large quantity of water and poured through a fine-meshed sieve. The coarser particles are retained and can be examined. A double bag of netting may be arranged upon a stout wire ring like a landing net and placed in the bowl of the water-closet. The fæces may be washed by repeated flushing and the retained particles examined for calculi. If they are not at first found every stool should be examined for several days, as they may be retained in the intestine for some time. Force should not be used in the examination, since recently formed biliary calculi are soft and may readily be disintegrated in handling them. The seeds of various fruits, particles of bone, and small fecal concretions are sometimes brought to the physician as gall-stones, and the rounded saponaceous masses voided after the ingestion of large quantities of olive oil are frequently mistaken for them, but these substances never contain cholesterin or bile pigment in quantity, nor do they present the internal structure of gall-stones.

Differential Diagnosis.—In *right-sided renal colic* the pain begins in the lumbar region and radiates toward the groin. There is retraction of the testicle and pain in the glans penis. Jaundice, tenderness in the region of the gall-bladder, and fever are not usually present. A calculus may be voided by way of the urethra. *Peptic ulcer* may suggest biliary colic. The pain, however, usually follows the ingestion of food and is burn-

ing in character, passing to the back. The vomiting is less urgent and the vomitus may contain blood. There is localized epigastric tenderness and anæmia. *Nervous hepatic colic*—the pseudobiliary colic of nervous women—may lead to an erroneous diagnosis. The pain is referred to the right side and may radiate to the back or shoulder. It is dull and dragging rather than colicky. The attack follows emotional excitement or fatigue. There may be tenderness upon pressure, but jaundice does not occur. *Intestinal colic* is relieved by belching, the passage of flatus, or defecation. It is more generalized and less intense than biliary colic and not followed by jaundice. *Lead colic* may simulate gall-stone colic, but the occupation of the patient is suggestive, while stubborn constipation, the gingival line, wrist-drop, hard arteries, and albuminuria constitute a characteristic symptom-complex. Jaundice is absent.

3. **The Symptoms Caused by Permanent Obstruction of the Ducts by Gall-stones.**—The obstruction may involve the cystic duct, the common duct, or the hepatic ducts.

I. Obstruction of the Cystic Duct.—Occlusion of the cystic duct by a calculus or by the contraction of a cicatrix following ulceration does not always cause serious symptoms. It is liable to be followed by, (a) *dropsy of the gall-bladder—hydrops vesicæ felleæ.* The tumor is cystic and gourd-shaped or pear-shaped, its narrow extremity being at its connection with the liver. The contents in recent cases are bile mixed with mucus or mucopus,—in older cases a clear, thin mucus containing albumin and of variable reaction to litmus paper. The tumor projects downward and may attain large dimensions. It is freely movable from side to side, unless fixed by adhesions, and when pushed backward turns to its original position as soon as the pressure is withdrawn. When the belly wall is thin and relaxed the outline of the distended gall-bladder may be visible, fluctuation may be elicited upon light bimanual percussion and palpation, and when there are many calculi present gall-stone crepitus may be felt. (b) *Atrophy of the Gall-bladder.*—This condition frequently follows dropsy of the gall-bladder. The contents undergo gradual resorption and the bladder contracts around any stones that it may contain, or, in the absence of a stone, into a small fibrous mass, or there may be diverticula in which calculi are embedded. In old cases of this kind lime salts are sometimes deposited upon the mucosa or in the bladder wall. (c) *Acute cholecystitis,* usually simple *but in rare cases phlegmonous.* (d) *Suppurative cholecystitis*—empyema of the gall-bladder. The gall-bladder may be greatly enlarged and contain as much as a litre of pus. Perforation may take place into the peritoneal cavity; more commonly adhesions take place with abscess formation.

The occurrence of these conditions may constitute the first direct evidence of cholelithiasis. Under no circumstances should exploratory puncture be performed. Aspiration has been followed by fatal results.

II. Obstruction of the Common Duct.—The duct may be occluded by a single stone in the ampulla of Vater or in any part of its course or by a number of stones which may also extend into the cystic and hepatic ducts. The obstruction may be, (a) *complete.* The calculus is tightly impacted in the common duct, or a large stone in the cystic duct compresses

the common duct at its upper part or the hepatic duct. There is complete bile stasis with deep and persistent jaundice and without septic phenomena. The common duct behind the obstruction, and the cystic and hepatic ducts may be enormously dilated and simulate the gall-bladder, for which they have been mistaken. The condition cannot always be differentiated from compression of the duct by new growths, though pain, a history of biliary colic, and absence of dilatation of the gall-bladder are in favor of a diagnosis of complete obstruction by gall-stones. Or, (b) *incomplete*. In this form there is cholangitis, which may be simple or suppurative. (*a*) Incomplete obstruction with non-suppurative cholangitis. There may be a single movable calculus in the diverticulum of Vater or in the duct above it,—ball-valve mechanism,—or a small faceted stone partially impacted, or a series of stones. The ducts above the obstruction are dilated, but the gall-bladder is often contracted. There are variations in the degree of jaundice and in the amount of bile pigment in the fæces. The liver is only slightly enlarged and the gall-bladder, as a rule, not at all distended. Finally there are irregular attacks of fever accompanied with demonstrable enlargement of the spleen. In well-marked cases of ball-valve calculus the paroxysms of fever are irregularly recurrent and resemble attacks of ague. They are characterized by remarkable rises of temperature,—103°–106° F. (39.5°–41.1° C.),—intense chills, profuse sweating, gastric disturbances and hepatic tenderness and pain. The jaundice is variable and often intense. The resemblance to malaria is superficial, the periodicity not being regular, the blood parasite not present, and quinine useless. This fever is known as hepatic fever or the hepatic intermittent fever of Charcot. The attacks in many instances recur after irregular intervals, during which the temperature is normal, for many months.

Courvoisier's Law.—In the great majority of cases of obstruction of the common duct by gall-stone the gall-bladder is contracted; in the majority of cases of obstruction from other causes the gall-bladder is dilated.

(*β*) Incomplete obstruction with suppurative cholangitis. The ducts are invaded by pyogenic organisms. The suppurative inflammation may extend to the intrahepatic ducts—diffuse intrahepatic cholangitis—and to the gall-bladder—empyema. Abscess of the liver and perforation of the gall-bladder with abscess formation may occur. There are septic phenomena. The liver is enlarged and tender; jaundice is of moderate intensity and persistent and there is fever of intermittent or remittent type. The course of the disease is comparatively short and the termination fatal. This is by no means rare as a terminal condition in old cases of cholelithiasis.

In cholelithiasis of the common duct there is frequently an associated catarrhal or interstitial pancreatitis and Cammidge's test may show characteristic crystals.

4. **Ulcerative Lesions Caused by Gall-stones.**—Biliary fistulæ are far from uncommon. Ulceration of the bile passages may occur without symptoms. As a rule, however, they tend to grave derangements of health. By the erosion of arterial branches in the course of the formation of fistulous tracts in various directions, they may cause hemorrhages which may be latent or manifest in the stools or vomit. In rare instances gall-stones

have perforated into the portal vein. Much more common are fistulous communications with the intestinal tract. The stomach is involved comparatively rarely, the duodenum frequently, the small intestine much less commonly, while fistulous communications with the colon have been occasionally encountered. There are instances of fistulæ involving the ureters, with the passage of stones into the bladder and of the direct passage of biliary calculi into the urinary bladder. Perforation into the pleura and into the lung may also occur. Cutaneous fistulæ of spontaneous origin are very uncommon, though they are by no means rare after operation. They usually open in the region of the fundus of the gall-bladder, but may appear near the umbilicus or above the pubes. The formation of these ulcerative tracts is always preceded by adhesions between the viscera directly involved and followed by the discharge of biliary calculi into the distant organ. The tracts themselves are often long and tortuous and sometimes there are diverticula containing gall-stones. Abscess formation is common.

5. **Gall-stones in the Intestines.**—When by way of the common duct, as sometimes may occur, or by a fistulous tract, a large gall-stone finds its way into the gut, it may cause intestinal obstruction, either directly or by ulceration followed by cicatricial contraction. The obstruction may occur at the pylorus and cause symptoms suggestive of carcinoma. More commonly it is in the lower part of the ileum. In the region of the cæcum the condition may simulate appendicitis. A small stone may enter the appendix or a diverticulum. A stone in the colon may sometimes be recognized upon rectal examination.

Diagnosis.—Cholelithiasis is recognized during life in a small proportion of the cases only. The direct diagnosis while the gall-stones remain quiescent in the gall-bladder may, in rare instances, be made by the discovery of a tumor in the region of the gall-bladder in which gall-stones may be felt. The walls of the bladder may be thickened by chronic inflammation or the seat of nodular carcinomatous growths. The differential diagnosis between an enlarged gall-bladder and a tumor of the kidney depends upon the fact that the former may be pushed backward into the abdomen, but directly returns to its former position, while the latter will remain in the position into which it is replaced.

A thorough röntgenological examination in accordance with recent methods reveals the presence of biliary calculi in about fifty per cent. of the cases. Negative findings are inconclusive. All available methods must then be employed.

The attack of colic is commonly the first positive sign of gall-stone disease. The symptoms of biliary colic and the differential diagnosis between that affection and others which resemble it have already been discussed. The discovery of gall-stones in the stools constitutes a positive diagnostic sign.

Exploratory puncture of the gall-bladder is attended with the danger of the escape of some of the contained fluid into the peritoneum and a general peritonitis. It is therefore to be emphatically condemned. An exploratory operation by incision is, on the other hand, comparatively safe and wholly justifiable in the presence of obscure and dangerous symptoms.

Prognosis.—The fact that in a large majority of the cases no symptoms occur, and that the diagnosis is so often simply a post-mortem finding, justifies the assertion that cholelithiasis is a benign disease. Although it is so often latent it is not, however, free from danger. On the contrary, the migration of the stones by way of the natural passages and, to a greater extent, by artificial channels caused by ulceration and adhesions may be attended by great suffering and serious risk of life. Even under these circumstances the outlook is not wholly unfavorable. When infection of the bile passages and gall-bladder has taken place with septic fever, especially when empyema of the gall-bladder, suppurative cholangitis, or abscess of the liver has occurred as a complication, the prognosis is highly unfavorable. The general recognition of the fact that cholelithiasis is a surgical disease has rendered the prognosis far more favorable than it was at a period when the sole dependence was upon drugs and mineral waters.

vii. Affections of the Blood-vessels of the Liver.

1. **Anæmia.**—Nothing is known of anæmia of the liver as a clinical condition.

2. **Hyperæmia.**—Two forms are to be considered, active and passive.

(a) **Active Hyperæmia; Congestion of the Liver.**—The liver is one of the most vascular organs of the body. The rapid influx of blood by way of the portal vessels during digestion brings about a physiological hyperæmia which is transient, but which in the cases of persistent overindulgence in the pleasures of the table may become the cause of functional or even of organic changes. Excessive quantities of food, strong spices, coffee, and especially alcohol are credited with causing hyperæmia of the liver, a condition to which sedentary habits also contribute. The condition thus caused constitutes a conspicuous feature in general plethora and obesity, and is frequently associated with gout, gravel, and glycosuria. Toxic agencies such as are present in the acute fevers and malaria, and, indirectly, the mode of life common among Europeans in tropical climates are causal factors.

Symptoms.—The manifestations of active hyperæmia of the liver are indefinite and rarely present alone. They consist of sensations of pressure and fulness in the right hypochondrium and epigastrium, which may amount to actual pain and are sometimes intensified by movement, deep respiration, and pressure. They are associated with the evidences of enlargement of the organ and certain symptoms of deranged digestion, such as epigastric weight, heart-burn, flatulence, and fulness in the head. In some instances a periodical recurrence of this group of morbid phenomena accompanies the return of menstruation or the suppression of the flow, or the arrest of an habitual hemorrhoidal flux. A slight icterus frequently accompanies the attack of hyperæmia of the liver. The tendency to the development of organic disease as a result of permanent hyperæmia is to be considered.

(b) **Passive Congestion.**—This form of hyperæmia is much more common. All conditions which favor the accumulation of blood in the venous system and the transference of blood-pressure from the arterial to the venous side of the circulation lead to passive hyperæmia of the liver, which

constitutes, in fact, an important part of the general visceral congestion characteristic of such states. Cardiac affections, both valvular and myocardial, especially when the power of the right ventricle is diminished, are important etiological factors. Pulmonary diseases which cause mechanical interference with the pulmonary circulation, such as acute and chronic bronchitis, emphysema, consolidation of the lung from exudate or sclerosis, tumors, and extensive pleural effusion or adhesions, also give rise to passive hepatic congestion, partly by reducing the calibre of the pulmonary vessels and partly by restricting the respiratory excursus. Deformities of the spinal column may act in the same way. Local hyperæmia is seen in the corset liver in the part separated from the main organ. The liver is enlarged, dense, and of a deep brownish-red color—the cyanotic or cardiac liver. Later it may be contracted, owing to the overgrowth of connective tissue.

Symptoms.—In the early stages symptoms are absent or subordinated to those of the general condition. Later epigastric fulness, especially after eating, dyspeptic disturbances and hemorrhoids occur. Hæmatemesis may occur. The portal obstruction may cause ascites. A mild jaundice is common. The stools may be clay-colored, and bile pigments may be present in the urine. Physical examination shows the liver to be enlarged. It is usually tender, especially below the arch of the ribs or upon pressure with the flat hand. Liver pulsation is often present.

Diagnosis.—The passive hyperæmic liver varies in size, whereas the enlargement of the liver from other lesions is persistent. This fact, considered in connection with the various etiological factors above enumerated, has great diagnostic significance.

3. **Diseases of the Portal Vein.** — (a) Occlusion or narrowing of the portal vein may be caused by, (1) acute or chronic inflammation of the vessel wall or by its invasion by a neoplasm; (2) compression from without by tumors, enlarged lymph-glands, gall-stones, or the cicatricial contraction of the adjacent parts, especially such as result from syphilis and tuberculosis; (3) mechanical obstruction caused by Distoma hæmatobium; and finally (4) from other causes not clearly understood. (b) Thrombosis.—The foregoing conditions cause slowing of the blood stream in the portal vein and thus favor thrombus formation, which may occur in cirrhosis, syphilis of the liver, malignant growths involving the wall of the vein, hyperplasia of the lymph-glands in the porta, compression or perforation of the walls of the vessel by hepatic calculi, parasite invasion, and arteriosclerosis. The coagulation forms a wall-thrombus, which may partially or wholly occlude the vein. (c) Adhesive Pylephlebitis.—In rare instances a collateral circulation is established, the thrombus becomes thoroughly organized, and the vein is converted into a fibrous cord.

Symptoms.—The condition manifests itself by acute symptoms which may supervene in the course of hepatic cirrhosis, chronic peritonitis, or abdominal tumor, or occur suddenly in persons apparently well. These symptoms consist of sudden intense epigastric pain with hæmatemesis, melæna, followed, in the course of a few days, by ascites and enlargement of the spleen. Icterus is sometimes present.

(d) Suppurative Pylephlebitis.—This condition is due to infection by pyogenic bacteria. It is secondary to intestinal ulceration such as

occurs in dysentery, enteric fever, or tuberculosis, to suppurative foci, as appendicitis, pelvic abscesses, ischiorectal abscess, inflamed hemorrhoids, or fistula. Multiple abscess formation occurs within the branches of the portal vein. Septic phenomena are rapidly developed. They consist of irregular chills, fever of rapid and extreme oscillation of temperature, colliquative sweating, and profound asthenia. Local symptoms are not constant. There may be tenderness and pain over the liver and spleen, the latter being enlarged. Icterus is not usually marked.

4. **Diseases of the Hepatic Artery.**—(a) Dilatation occurs in cirrhosis of the liver. (b) Sclerotic changes in the wall are common. These conditions are of pathological rather than of clinical interest. (c) Aneurism is infrequent. A number of cases have been studied clinically. The diagnosis is obscure. Important symptoms are pain, hemorrhage from the bowel and uterus. A pulsating tumor has never been found. The cases closely simulate gall-stone disease or duodenal ulcer. The combination of the symptoms of these two conditions is of diagnostic significance. Death may occur suddenly from hemorrhage into the gastro-intestinal tract or into the peritoneum.

5. **Diseases of the Hepatic Veins.**—(a) Dilatation occurs when the right heart is permanently dilated and hypertrophied. (b) Stenosis is far less common. It may be due to, (1) compression by tumors, especially gummata and enlarged lymph-nodes; (2) disease of the vessel walls; (3) thrombosis; and (4) embolism. The symptoms are obscure and the clinical diagnosis is uncertain. The spleen is palpable, the liver enlarged, and ascites occurs. (c) Thrombosis may occur as a result of stenosis. (d) Emboli may pass into the hepatic veins from the right auricle when the blood current is reversed, as may occur in tricuspid insufficiency. The subsequent course of embolism and thrombosis of the hepatic veins varies according to the nature of the cause of the occlusion, namely, whether it be simple, infected, or the result of malignant disease. Infarcts are occasionally encountered.

viii. Abscess of the Liver—Suppurative Hepatitis.

Definition.—Suppuration within the liver, either in the parenchyma or in connection with the blood-vessels or bile-passages.

The following forms occur: (1) solitary abscess, (2) embolic or pyæmic abscesses, (3) suppurative pylephlebitis, (4) suppurative cholangitis, (5) local abscess formation caused by foreign bodies or parasites.

The distinction sometimes made between primary and secondary abscesses of the liver cannot always be observed. Those abscesses due to traumatism or the extension of suppurative processes from the gall-bladder or bile-ducts are primary; those in which infection has taken place by way of the blood stream are secondary.

Etiology.—Predisposing Influences.—Residence in the tropics, especially when associated with excesses at table and overindulgence in alcohol, amœbic dysentery, cholelithiasis, appendicitis, traumatism in the region of the liver and blows upon the head, ulceration of the intestines and suppurative processes in the pelvis, general sepsis with metastatic abscesses, and echinococcus cysts in the liver are all predisposing factors of importance.

The Exciting Cause.—Infection of the liver substance, the blood-vessels, the bile-ducts, or the gall-bladder by pyogenic organisms is the direct cause of hepatic abscess. The avenues of infection are the portal vein, the hepatic vessels, the common duct, and penetrating wounds or fistulous tracts. The organisms found in hepatic abscess comprise Amœbæ dysenteriæ, streptococci, colon bacilli, pneumococci, typhoid bacilli, Bacillus pyogenes, and actinomyces.

Morbid Anatomy.—Tropical abscess is usually solitary, though two or more abscesses are occasionally encountered. When single, the abscess is commonly situated in the right lobe, extending to the upper surface, less frequently toward the concave surface of the organ. Recent abscesses, as seen at the surface of the liver, are of a grayish-yellow color with a well-defined outline. In a more advanced stage the walls are shreddy and necrotic and contain a greenish- or reddish-brown viscid pus commingled with blood, in which there are fragments of liver tissue. This fluid shows fatty and granular detritus, cellular elements, occasionally Charcot-Leyden crystals, and amœbæ, which are also present in the tissue forming the wall. Cultures are commonly sterile. The abscess wall consists of an inner necrotic layer, a middle layer in which there is proliferation of connective tissue, and an outer layer of intense hyperæmic tissue. The chronic abscesses frequently have an extremely dense wall. Rupture may occur into the lower lobe of the right lung, into the pleura (causing an empyema), into the vena cava, the portal or hepatic veins, or into the stomach, intestine, peritoneum, pericardium, or externally. Perforation into the right kidney is a rare event. Traumatic abscess is usually single and has no distinctive anatomical characters. Pyæmic abscesses are almost always multiple and may be very numerous. When large they are due to the coalescence of several small cavities. The infected emboli find their way to the liver through the hepatic artery or form infected thrombi in the portal vein. Echinococcus cysts undergo suppurative changes as the result of infection following trauma or inflammation of the bile passages. The abscesses may be of great size, and contracted hydatids, dead scolices, shreds of the cyst wall, fat, and bilirubin may be found in the pus.

Symptoms.—1. Solitary Abscess.—This condition is common among Europeans in the tropics and chiefly affects males. It occurs also in the temperate zones. It is almost always associated with amœbic dysentery, though cases occur in which no history of dysentery can be obtained. The abscess may present no definite symptoms and finally reveal its presence by rupture which may prove fatal. The principal symptoms are fever, pain, tenderness, and septic phenomena. Enlargement of the liver may be demonstrated. The temperature is irregular. There is paroxysmal fever of intermittent type, sometimes of regular, sometimes of irregular, periodicity, the temperature frequently falling to subnormal ranges. Rigors are followed by rises of temperature to 103°–105° F. (39.5°–40.5°C.), followed by copious sweating. The condition frequently simulates a malarial intermittent fever. In the chronic cases fever may be absent. Pain referred to the region of the liver, the back, the epigastrium, or the right shoulder is a common symptom. It is often associated with sensations of weight and dragging in the right hypochondrium, much increased

when the patient turns upon his left side, and with tenderness upon pressure at the costal margin in the mammillary line. The shoulder pain is due to irritation of the terminal filaments of the right phrenic nerve which are distributed to the capsule of the liver. The stimulus is transmitted to the fourth cervical, which anastomoses with the phrenic and sends sensory branches to the shoulder. The facies of the patient is sallow, pallid, muddy, and faintly icteroid. Irregular diarrhœa, digestive disturbances, and great mental depression are common. Leucocytosis is sometimes present, often absent, and therefore not usually of diagnostic value. Enlargement of the liver is most marked in the right lobe and upward. This condition is in contrast with many diseases of the liver in which the enlargement is in a downward direction, as fat infiltration, hypertrophic cirrhosis, and carcinoma. The upper margin of liver dulness is usually higher in the back near the spine than anteriorly. In extensive abscess formation the lower margin of the liver may extend a hand's breadth below the edge of the ribs. The entire right hypochondrium may bulge, the ribs protrude and be widely separated, and fluctuation may be detected. Palpation may be painful. The margin of the liver is felt to be rounded and blunt, the superficial venules over the liver may be dilated, and there may be circumscribed cyanosis with œdema. Upon deep respiration a friction fremitus may sometimes be detected. Perforation into the lung may occur or amœbic infection through the diaphragm without rupture. The base of the right lower lobe shows signs of consolidation, there is intense paroxysmal cough, with characteristic expectoration resembling anchovy sauce and containing Amœbæ coli in varying numbers and actively motile. The color of the expectoration is due to altered blood. The sputum may be blood-tinged or bright red and very abundant. It may contain pus and shreds of liver tissue. Recovery may take place in the course of several weeks. Perforation into the pleura causes empyema with characteristic symptoms and physical signs; into the pericardium is followed by fatal collapse; into the stomach gives rise to vomiting of blood-tinged fetid pus; into the intestine to the presence of pus in the stools. Coincident with the escape of pus in these various directions there is sudden subsidence of the liver tumor, together with sensations of collapse. Rupture into the vena cava is followed by rapid death with symptoms of asphyxia. In the rare cases of perforation of a liver abscess into the pelvis of the right kidney, the urine contains pus of a brownish-red color which may show the presence of liver-cells or blood-corpuscles.

2. Embolic or Pyæmic Abscesses.—The multiple small abscesses in the liver, which occur in some cases of general septicopyæmia, may cause pain and tenderness in the hepatic region and a slight subicteroid discoloration of the skin. When these symptoms are superadded to the rigors, high temperature, sweating, and prostration of the septic condition, and especially when a suppurative focus can be discovered, the diagnosis of metastatic abscesses in the liver may be made.

3. Suppurative Pylephlebitis.—The clinical manifestations are the same as in pyæmic abscess. The liver is enlarged and tender, there is fever of septic type, and a muddy, icteroid skin.

4. Suppurative Cholangitis.—The history of attacks of gall-stone colic or of the recovery of gall-stones from the stools, and the symptoms of cholangitis or the presence of a distended gall-bladder are important for the diagnosis.

5. Foreign Bodies and Parasites.—Needles have penetrated the wall of the œsophagus or stomach and entered the liver substance, causing abscess. A needle or a fish-bone has been known to perforate a branch of the portal vein and give rise to pylephlebitis. The part played by echinococcus cysts in producing liver abscess has been described. The penetration of round worms into the common duct and, less commonly, the presence of flukes have caused suppurative processes. These parasites probably act merely as carriers of pyogenic germs.

Diagnosis.—Direct.—Abscess of the liver is often latent and wholly overlooked, notwithstanding the fact that the patients almost always present the appearance of serious illness. This is due to the fact that local symptoms may be altogether absent or subordinated to those of the primary disease. When pain, tenderness, and enlargement of the liver are present, and a source of infection can be found in the structures tributary to the portal vein; or a focus of purulent inflammation elsewhere; or when there is a history of traumatism involving the liver, or of bone injury, especially injury to the bones of the skull, or of cholelithiasis, a positive diagnosis may be made. Nevertheless the symptoms are frequently obscure, and the diagnosis, even when abscess is suspected, cannot in many cases be positively determined. This statement is especially true of abscesses of moderate size centrally situated in the substance of the liver, and of the multiple small abscesses which occur in general septic conditions and in acute cholangitis and pylephlebitis. Of positive diagnostic import are pain referred to the region of the liver and the right shoulder, enlargement of the liver in an upward direction, bulging, particularly when circumscribed, and fluctuation. Examination by the X-rays yields, as a rule, unsatisfactory results, owing to the density of the liver, but in exceptional cases may be of service.

Differential. — The following conditions are to be considered: (a) *Malarial Fever.*—The regularly intermittent fever often closely simulates malarial fever, but the absence of marked splenic enlargement and of the malarial blood parasite, and the failure of quinine to control the fever are of diagnostic significance. (b) *Right-sided Empyema.*—When the abscess ruptures into the pleura an empyema is produced, but perforation of the lung commonly follows, and the true nature of the condition is revealed upon the expectoration of pus resembling anchovy sauce and containing amœbæ. (c) *Subphrenic Abscess.*—The downward displacement of the liver, the fact that the lower border of the lung descends upon deep inspiration, and a history of gastric ulcer may be of service in the differentiation from hepatic abscess. When gas is also present,—pyopneumothorax subphrenicus,—the diagnosis is less difficult. (d) *Abscess of the Abdominal Wall.*—Perforation through the abdominal wall presents little difficulty. A mural abscess may closely simulate liver abscess. Such lesions, usually associated with tuberculosis of the ribs, are superficially situated, and an exploratory needle does not follow the movements of

respiration. The liver is not enlarged. (e) *Empyema of the Gall-bladder.*—The situation of the tumor, its gourd-like outline, and some degree of lateral movement upon pressure are important points in diagnosis. (f) *Echinococcus Cysts.*—Slowness of growth, absence of fever and signs of inflammation, very obscure fluctuation, and the hydatid thrill are characteristic of these cysts. When they become infected the differential diagnosis from hepatic abscess is attended with great difficulty. The presence of hooklets or shreds of cyst walls in the pus is of positive diagnostic significance. (g) *Abscess of the Left Lobe.*—An abscess in this position is uncommon. It may, in the absence of inflammatory symptoms, simulate carcinoma ventriculi, from which it may be distinguished by the age of the patient, the history of the case, an examination of the gastric contents, and the presence of foci of infection in the intestines or elsewhere. (h) *Aneurism of the Aorta.*—Hepatic abscess to which the movements of the aorta are transmitted may simulate an aneurism in the region of the cœliac axis. In abscess the pulsation is to and fro, not expansile, diastolic shock is absent, a bruit is not heard, and the pain is less severe and paroxysmal than in aneurism and has a different focus of maximum intensity. (i) *Hepatic Fever.*—The intense fever with chills and sweating which occurs in certain cases of gall-stone disease is frequently regarded as due to abscess of the liver. It is now known that this symptom may occur in the absence of suppuration. The fever occurs in paroxysms, which may have a regular periodicity or recur at irregular, often prolonged, intervals, during which there is complete apyrexia and the nutrition may be fairly well maintained. The varying jaundice, which is intensified during the febrile attacks, is suggestive, and the long duration of the condition is of diagnostic importance.

Exploratory puncture may be made in a doubtful case. The patient must be etherized and the skin cleansed as in any surgical operation. The aspirator needle, which should be of large calibre, should be introduced at the suspected point in the infra-axillary region or over the area of hepatic dulness behind. Repeated puncture may be necessary.

Prognosis.—The outlook in pyæmic abscesses is ominous. In diffuse suppurative cholangitis and pylephlebitis and in traumatic abscess it is highly unfavorable. In solitary, tropical abscess it is much more hopeful. Recovery may take place after aspiration or incision. In the more chronic cases of solitary abscess, perforation into the lung or the intestine or through the skin may be followed by recovery. In individual cases marked septic phenomena, persistence of dysenteric symptoms or of suppuration in pelvic abscess or bone disease, the supervention of amyloid disease, and the development of cachexia are highly unfavorable. The danger of rupture into the vena cava, the pericardium, or the peritoneum renders the prognosis uncertain.

ix. Fatty Liver.

Definition.—This term is used to designate all conditions characterized by an abnormal increase in the fat of the liver.

Under physiological conditions the fat in the liver-cells varies and is dependent upon the amount and character of the food. The ingestion

of large quantities of fat is followed by an increase of fat-globules in the peripheral cells of the acini. This increase is transient, disappearing in the course of several hours.

Pathologically two different forms of fatty liver occur—fatty infiltration and fatty degeneration. These forms sometimes coexist. Fatty infiltration is the result of an increased deposit of fat in the parenchyma of the liver in the absence of fatty change in the protoplasm of the cells.

Fatty degeneration consists of a destruction of the protoplasm of the cells with fat accumulation.

Etiology.—Fatty infiltration occurs in general obesity and in persons who habitually consume inordinate amounts of fats and carbohydrates; at the middle periods of life; and in many women after the menopause. It is common in conditions in which there is deficient oxidation, marked anæmia, advanced phthisis, or the cachexias. Chronic alcoholism leads to fatty degeneration of the liver by interfering with the oxygenation of fats and carbohydrates, the oxygen being largely required for the combustion of the alcohol consumed. Fatty degeneration is caused by certain poisons, as phosphorus, the toxin of acute yellow atrophy, and arsenic, mercury, and antimony.

Morbid Anatomy.—The ordinary fatty liver—fatty infiltration—is uniformly enlarged and may reach double the normal weight. It is smooth, of a pale yellow or drab color, and greasy. In fatty cirrhosis the surface shows irregular granular prominences. On section the light yellow color and empty blood-vessels are noticeable and the knife is smeared with grayish-white fat. Microscopically the cells are distended with small and large fat droplets.

Symptoms.—Clinically fatty infiltration is to be distinguished from fatty degeneration. In the former the parenchyma is not degenerated but simply contains an excess of fat. The hepatic functions are therefore maintained, and general symptoms do not occur. In the latter the liver-cells are affected by a degenerative process, their functions are no longer performed normally, and grave symptoms are present, as in acute yellow atrophy, q.v., and phosphorus poisoning. The symptoms in fatty infiltration are not well defined. The appearance of the patient and the symptoms referable to other organs are dependent upon the primary disease. Jaundice does not occur. The stools may be light-colored or even whitish-gray, and putty-like in consistency. The urine does not contain bile pigments in excess. Hemorrhoids may occur, but the signs of portal obstruction are not common. In obese persons the physical signs are often obscure, but in phthisis and cachectic conditions the greatly enlarged liver may be sometimes visible through the belly wall and almost always recognized upon palpation. It is smooth, with a rounded lower border, projecting far below the margin of the ribs, and is painless.

Diagnosis.—Slight grades of fatty liver cannot be recognized during life. When the condition is well developed it is, except in the case of great obesity, easy of recognition. The great enlargement, smooth surface, and nearly normal contour are characteristic. The underlying disease, as anæmia, phthisis, the various cachexias, is of diagnostic importance. The soft consistence enables us to exclude amyloid liver, leukæmic tumors, and hyperæmia.

Prognosis.—Fatty liver is a secondary pathological condition, and the prognosis is that of the primary disease.

x. Chronic Interstitial Hepatitis.

Cirrhosis of the Liver.

Definition.—A chronic disease of the liver due to various toxic or infectious causes, mechanical irritation, or stasis of blood or bile, and characterized by an overgrowth of the interstitial connective tissue of the organ.

It is of interest to investigate the etymological significance of this unfortunate word "cirrhosis," originally selected by Laennec to describe an anatomical peculiarity of the contracted liver far from constant, and subsequently applied to changes in other viscera attended by an overgrowth of connective tissue, so that there have been those who have spoken of cirrhosis of the kidney, cirrhosis of the lungs, and the like. The word is derived from the Greek *χιρρος*, tawny or orange-yellow, the color of the liver substance in some of the cases. It has nothing whatever to do, save by a remote and misleading association, with the sclerotic changes due to hyperplasia of connective-tissue stroma.

The difficulties in the diagnosis of hepatic cirrhosis are not, as in most other diseases, accidental. They do not arise from the peculiarities of individual cases. They are essential and due to the fact that no definition can be framed that at once pathologically and clinically includes all the cases. This arises from Laennec's choice of a term descriptive of an inconstant phenomenon and without pathological significance. "Cirrhosis" is conspicuous among the nosological terms that hamper medicine and obstruct the progress of knowledge. If it could be erased from the list of diseases our conception of the various conditions to which it is applied would be greatly simplified. The one underlying lesion common to all of them is an overgrowth of the fibrous tissue of the liver. The term chronic interstitial hepatitis is at once descriptive of the anatomicopathological condition and sufficiently comprehensive to include all the cases. Its more general use is to be desired.

Etiology.—The etiological relations of individual cases may be indicated by qualifying adjectives:

(a) In general toxic, and in particular alcoholic, plumbic, gouty, diabetic, rachitic, and the like. We must include here forms of interstitial hepatitis due to chronic phosphorus poisoning and the abuse of condiments. The assumption that intestinal autointoxication gives rise to interstitial hepatitis requires confirmation.

(b) In general infectious; in particular the specific febrile infections are occasionally followed by chronic interstitial hepatitis. Malaria produces similar changes, and syphilis gives rise to three well-characterized forms, namely, diffuse syphilitic hepatitis, commonly congenital, gummata which undergo fibroid transformation, and an extensive perihepatitis with increase in connective tissue of the portal canals.

(c) Mechanical irritation. The long-continued exposure to an atmosphere laden with dust particles such as produce pneumonoconiosis, whether these be mineral or metallic, may also give rise to an inflammatory process in the connective tissue of the liver.

(d) Congestive; the chronic hyperæmia of the blood-vessels occurring in heart disease gives rise to an interstitial hepatitis—the cardiac liver.

(e) Obstructive, the result of chronic obstruction in the bile-ducts.

Pathology and Classification.—Vaughan, in a recent study of the subject, concludes that so-called atrophic and hypertrophic cirrhosis are not different forms of the same disease but wholly different diseases. He states that the former is known as atrophic cirrhosis because from its earliest possible recognition the liver is less than normal in size; while the hypertrophic is known as such because at every stage of the disease the liver is larger than normal; that the atrophic is known as venous cirrhosis because of the early and constant involvement of the intrahepatic branches of the portal vein, the hypertrophic as biliary cirrhosis because of the early appearance and constancy of icterus; that in atrophic cirrhosis the primary destructive changes are in the hepatic cells, while in hypertrophic cirrhosis the epithelium of the gall-ducts is the site of the primary involvement; that the former might be known as toxic, the latter as infective cirrhosis.

This clear-cut classification, while in some respects convenient and while it provides categories for well-marked cases of widely different morbid conditions, by no means meets the requirements of all the cases and does not appear to be wholly justified upon etiological, anatomical, or clinical grounds. There are cases of atrophic cirrhosis in the causation of which alcohol plays no part, and cases of hypertrophic cirrhosis in hard drinkers. My clinical experience leads me to believe that alcohol is a very common cause of the latter form of cirrhosis.

Cases of cirrhosis of the liver without jaundice and with the evidences of a high degree of portal obstruction, in which the liver is normal in size or slightly enlarged, are by no means rare. In addition to these there is the fatty cirrhosis common in beer-drinkers. There is a large group of cases in which the symptoms are neither those of the atrophic nor the hypertrophic form. These are designated mixed forms, and are thought to be due to the coexistence of the two forms, the toxic and the infective, in the same individual. On the other hand, Hawkins of St. Thomas's Hospital, in the article on cirrhosis of the liver in Allbutt's System, expresses the opinion that the terms "atrophic" and "hypertrophic" are scarcely worth retaining. "The former," he observes, "has lost much of its fitness now that statistics show that the hob-nailed liver, to which it was originally applied, is not necessarily small, but is often increased both in size and weight," while "the phrase hypertrophic cirrhosis has become so complicated by the postulate of a biliary cirrhosis that its significance is vague and uncertain." Hawkins does not recognize a vascular or toxic form corresponding to the atrophic form of writers—Laennec's cirrhosis—and a biliary or infectious form—Hanot's cirrhosis,— but states that "two forms of cirrhosis of the liver are induced by the excessive use of alcohol." These two forms are separated both in their morbid anatomy and in their clinical features. In the first, which is by far the more common, the newly developed fibrous tissue is "multilobular," that is, it tends to surround large groups of hepatic lobules. This form is commonly associated with ascites but seldom with jaundice. In the second and less common form

of *alcoholic cirrhosis* the new tissue is mostly developed around single lobules and the condition is properly described as "unilobular." There is little tendency to ascites, while jaundice is common.

In this connection may be mentioned Flexner's researches in regard to the new tissue in cirrhosis of the liver. He found both the white fibrous tissue and the elastic tissue increased and the chief distinction between the histology of the atrophic and hypertrophic cirrhosis to depend upon the degree of extralobular growth and the freedom with which the lobules are invaded, and that "in hypertrophic cirrhosis there would appear to be less interlobular growth and an earlier and finer intralobular growth."

Symptoms.—Clinically not all forms of interstitial hepatitis can be recognized. The symptoms are often vague and referable to other organs, especially those of the gastro-intestinal tract. In many of the cases the condition during life can only be suspected, the morbid phenomena being those of the primary disorder. Direct physical signs are available for diagnosis only when there are definite changes in the size or contour of the liver. Hence the cases of chronic interstitial hepatitis, whatever their course, must be arranged in three groups according as the liver is found upon physical exploration to be, (a) of about the normal size, (b) atrophic, and (c) hypertrophic.

(a) Since there are no characteristic symptoms in many cases of chronic interstitial hepatitis, and since in many of the cases the liver remains of normal size throughout and is always of normal size until the disease has made some progress, it follows that the diagnosis is impossible in a considerable proportion of the cases and in the earlier stages of all cases. The most that can be done is to assume that when certain etiological factors, as alcoholism, malaria, valvular disease of the heart with failing compensation, are operative, and more or less well-pronounced gastrohepatic symptoms present, the patient may have an interstitial hepatitis. The diagnosis under such circumstances must be purely an anatomical one, and this is frequently the case in the fatty form of interstitial hepatitis in which symptoms directly referable to the liver are often absent, and not rarely in the multilobular form either with or without atrophy. In the latter instance the true nature of the malady may reveal itself in sudden copious hæmatemesis or in the peculiar toxæmia caused by the entrance of portal blood, which has not traversed the liver, into the general circulation.

When, however, there are decided changes in the size of the liver the state of affairs is wholly different. The interstitial hepatitis reveals itself not only in physical signs, but also in symptoms of significance.

(b) The liver is diminished in size—*Laennec's cirrhosis.* A high degree of atrophy may occur, provided the collateral circulation has been established, without the development of ascites or other symptoms which attract the attention of the patient to the liver. As a general rule, however, the malnutrition, vomiting, the enlargement of the spleen, hemorrhoids, the distended superficial abdominal veins, and the increased girth caused by the ascites are diagnostic. It is of cardinal importance also to remember that all these phenomena can occur in an interstitial hepatitis in which the liver is not only not atrophic but even somewhat enlarged. Under these circumstances jaundice is not usually present.

(c) The liver is increased in size. The hypertrophic form of chronic interstitial hepatitis,—*Hanot's disease*,—like the forms already considered, may present extreme difficulties in diagnosis in its early stages. The liver may be as yet of normal size. The early icterus may resemble that of catarrhal jaundice—a resemblance heightened by its fluctuation or temporary disappearance, and by the occurrence of fever. The recurrence or persistence of the jaundice, its intensity, and the presence of bile in the stools are of diagnostic importance. When the disease is established the uniform enlargement of the liver, the splenic tumor, the deep and persistent jaundice, and the occasional attacks of fever are diagnostic. The fever is of subcontinuous or remittent type and extends over periods of days or weeks. Chills and sweating are not common, as in the fever of impacted gall-stone —Charcot's or true hepatic fever.

FIG. 314.—Ascites due to atrophic cirrhosis of the liver.—Jefferson Hospital.

Diagnosis.—DIRECT.—The diagnosis of well-defined cirrhosis of the liver, the terminal condition, is usually a simple matter. Whether it be the atrophic form of Laennec or the hypertrophic form of Hanot, the symptom-complex in most of the cases is characteristic. In the former the pinched face with its distended venules and muddy or subicteroid hue,—*facies hepatica*,—the spare chest and thin arms, the distended belly with its conspicuous superficial veins, and the diminished area of liver dulness leave no doubt of the nature of the malady. In the latter the jaundice, the fairly well-preserved nutrition, the big liver without ascites, and occasional irregular fever render the diagnosis equally clear. The two conditions are clinically distinct because they are the manifestations of essentially different pathological lesions. The one thing that these lesions have in common is an overgrowth of the interstitial connective tissue of the liver. It might be said that they have nothing in common clinically save that they are diseases of the liver. Yet they are described respectively as the atrophic form and the hypertrophic form of the same affection, hepatic cirrhosis.

The diagnosis of hepatic capsulitis—the *capsular cirrhosis* of authors—is usually attended by insuperable difficulties. The symptoms are those

of the atrophic form of chronic interstitial nephritis. Jaundice is not usually present. The kidneys are granular. This condition may be associated with perisplenitis and proliferative peritonitis.

Differential.—Adhesive pylephlebitis closely resembles the atrophic form of interstitial hepatitis. The etiological factors and the rapidity with which the peritoneal effusion develops and reforms after tapping, as was shown in a case recently in my service in the Pennsylvania Hospital, are important in the differential diagnosis. It is said that thrombosis of the portal vein is followed by an atrophy of the liver, which renders the diagnosis difficult. In the cases that have come under my observation death has occurred in the course of a few weeks, and the liver was of normal size.

Prognosis.—In the atrophic form the outlook is highly unfavorable. From the time when the diagnosis can be made the duration of the disease is often not more than a few months, rarely more than two or three years. Atrophic cirrhosis in advanced stages has been found post mortem in cases in which no characteristic symptoms were present during life. Life has been much prolonged in some of the successful cases of omentopexy.

In the hypertrophic form the disease, after it has reached a stage in which a positive diagnosis can be made, runs an unfavorable course. The progress is often slow and the disease for a long period may not continuously prevent the patient from conducting his business or taking part in the ordinary affairs of life.

xi. New Growths in the Liver.

Neoplasms of the liver are benign and malignant. The benign are fibroma and angioma; the malignant are carcinoma, sarcoma, and malignant adenoma. Carcinoma and sarcoma may be primary or secondary.

1. **Benign New Growths.**—These are of no great clinical importance.

(a) **Fibromata.** — These tumors consist of dense connective tissue and are frequently found post mortem. They are usually small and cause no symptoms during life. In very rare instances they are of larger size, and when so situated as to compress the bile passages and prevent the discharge of bile into the intestine they may cause cholangitis and death from cholæmia.

Diagnosis.—In a suspected case an exploratory laparotomy should be performed in the hope that the tumor may be found and removed.

(b) **Angiomata.**—These vascular tumors are also described under the terms cavernomata and telangiectasis. They are commonly multiple and of small size, causing no symptoms. They may be found at all ages and have been observed in the fœtus. In very rare instances they are solitary and of large size,—an orange,—the pregnant uterus. They then give rise to pressure symptoms.

Diagnosis.—Small angiomata cannot be recognized by clinical methods. In some instances the surface of the liver is nodular. A large tumor giving rise to distressing pressure symptoms may justify a diagnosis by exclusion. Its true nature can only be positively determined by an exploratory incision.

Prognosis.—The outlook is favorable as regards life. Large solitary angiomata have been successfully resected.

2. **Malignant Tumors of the Liver.** — Cancer of the gall-bladder and bile-ducts and its relation to cancer of the liver has already been considered.

Malignant tumors of the liver are of great clinical importance.

(a) **Carcinoma Hepatis.**—Carcinoma invades the liver less frequently than the uterus, the stomach, or the breast. Cancer of the liver may occur at any age and has been observed in the new-born. A number of cases of primary carcinoma of the liver have been observed in children. The part played by heredity in cancer of the liver is uncertain. About 17 per cent. of the cases show hereditary predisposition. Half the cases are first observed after the fortieth year. Primary cancer of the liver is more common in men and is frequently associated with cirrhosis. Secondary cancer is much more frequent in women, a fact attributed to the remarkable tendency to cancer of the uterus, ovaries, and breasts. Primary cancer is common in the gall-bladder, rare in the parenchyma of the organ. The frequent association of gall-stone disease with cancer of the gall-bladder and bile-ducts is attributed to the chronic irritation caused by the presence of calculi.

Primary carcinoma presents three principal types: (i) *Massive Carcinoma.*—The liver is greatly enlarged, its surface usually smooth. On section the growth is sharply contrasted with the normal tissue which surrounds it. It is grayish-white and not usually softened. It is of very great size and at first solitary, though later surrounded by smaller metastatic nodules. (ii) *Nodular Carcinoma.*—Round, grayish-white, or yellow nodules of cancerous tissue of varying size are irregularly scattered throughout the liver. The occasional occurrence of one large mass surrounded by numerous smaller and less dense nodules makes it probable that the former is the original seat of the disease. The liver is not usually greatly enlarged. Sclerotic changes are common and the organ may be reduced in size. Transitional forms between massive and nodular cancer occur. (iii) *Adenocarcinoma with Cirrhosis.*—The liver is usually contracted but may be enlarged. The surface is dark green, with irregularly distributed yellowish nodules of varying size beneath the capsule. The adenomata appear throughout the liver substance as small round masses varying in size from a millet-seed to a pea. Sometimes only a single tumor is present.

Secondary Carcinoma.—The liver may be enormously enlarged. Nodules are present upon its surface, which may often be felt and seen through the abdominal walls. They may be dense or soft, and frequently, in consequence of retraction of the connective tissue, are distinctly umbilicated—*Farre's tubercles.* The tumors are irregularly scattered throughout the substance of the organ. They are usually light in color, grayish-white or greenish, and sharply defined, both on the surface and in the interior of the liver, from the surrounding liver substance, which is often hyperæmic. The nodules themselves are often hemorrhagic. They may be present in such numbers as almost completely to replace the liver parenchyma.

Histologically the primary cancers are epitheliomata; the secondary cancers are of the same structure as the respective primary growths—as a rule, alveolar or cylindrical carcinomata.

(b) **Sarcoma.**—Sarcoma of the liver is much less common than carcinoma. Primary sarcoma is very rare. Most of the cases are secondary, though the primary growth may be so small as to be easily overlooked. Melanosarcoma is the most common and most important variety. It appears in rare cases as a primary tumor. As a rule, it is secondary to pigmented sarcoma of the eye or skin. The primary growth is of diagnostic importance. The secondary sarcomata of the liver sometimes form enormous tumors. They occur either as large nodular masses throughout the liver or as diffuse, infiltrating growths. In the latter form the enlargement of the liver is less marked. On section the surface is black or marbled. They constitute part of a widely extended metastatic process in which many viscera are often involved.

Symptoms of Malignant Disease of the Liver.—The condition may be latent, especially when the growth develops centrally or in the diaphragmatic surface of the liver, or the symptoms may be overshadowed by those of the primary tumor. When a primary cancerous growth in any organ, as the breast, stomach, or rectum, is present or has been removed, the secondary nature of a neoplasm in the liver is obvious. When no such growth is discoverable, it is not always possible to determine whether cancer of the liver is primary or secondary. Loss of appetite and nausea are early symptoms. Vomiting is less common. Emaciation and loss of strength are marked and progressive. The skin loses its elasticity and becomes dry, wrinkled, and of a muddy color. Sensations of fulness and weight in the epigastrium and right hypochondrium are followed by pain which extends to the chest and right shoulder. The liver is commonly enlarged, tender upon pressure, and nodular. In some cases of primary cancer, and in those cases in which cirrhosis is marked, the liver may be of normal size or even contracted. Persistent jaundice is present in more than half the cases. It is intense when there is compression of the common duct. Ascites is much less frequent. It occurs as the result of compression of the portal vein, or its invasion by the growth, advancing cirrhosis, or extension of the growth to the peritoneum. The anæmia is progressive. Œdema of the feet or general œdema occurs; there are signs of metastasis to the pleuræ and peritoneum. Fever of continuous or remittent type occurs and death results from asthenia. The blood shows the changes of secondary anæmia which may be of high grade. Poikilocytosis may be present. Profuse diarrhœa may cause concentration of the blood.

Physical Signs.—Inspection.—The abdomen is distended, especially in the epigastric zone and upon the right side. In advanced cases the nodular surface and even the umbilication of the nodules may be visible through the emaciated abdominal wall. The superficial veins are usually enlarged. Palpation.—The enlargement may be recognized and the border felt some distance below the margin of the ribs. When the left lobe is affected, a distinct tumor may be felt in the epigastrium. The surface is commonly irregularly nodular, with an uneven margin and distinct central depressions in many of the nodules. In cases of diffuse infiltration the surface is usually smooth. It is also hard. Enlargement of the superficial lymph-nodes, especially the inguinal, supraclavicular, and cervical nodules, is common. When slight it is not due to metastasis and

not of diagnostic importance. Actual metastasis with decided enlargement of the left supraclavicular gland occasionally occurs in malignant disease of the abdominal organs. PERCUSSION.—The upper and lower limits of dulness may be determined and the progress of the growth estimated, the presence and increase of ascites observed, and pleural and peritoneal new growths recognized. The spleen is not usually enlarged.

Diagnosis. — DIRECT. — Enlargement of the liver, which may be smooth or nodular, and in particular when the nodules are umbilicated; cachexia; compression symptoms in the territory of the portal vein or vena cava—ascites, œdema; in the territory of the bile passages—jaundice; signs of metastasis in the pleuræ, lungs, or peritoneum, constitute a characteristic symptom-complex. In the presence of a primary growth or the history of one removed, the direct diagnosis may be made. In the absence of such a primary growth the diagnosis remains uncertain in proportion as one or more of the above groups of clinical phenomena are ill defined or absent. The Röntgen rays may be of great service in the diagnosis of doubtful cases in which localized dense neoplasms exist in regions inaccessible to the ordinary means of clinical examination.

Sarcomata of the liver are mostly secondary growths. In melanosarcoma the primary growths are commonly in the choroid or the skin. The enlargement is rapid and reaches a high grade. Multiple tumors are often present in the skin, and metastases are widely extended. Melanuria is an inconstant but important symptom. The liver tumor may not develop until some months after the removal of an eyeball, as in a case of mine in the Philadelphia Hospital.

DIFFERENTIAL.—*Fatty Liver.*—The uniform enlargement and smooth surface are suggestive of cancerous infiltration, especially in the fatty liver of emaciation and cachexia. But the soft consistency of the enlarged liver, its slow growth, and the absence of jaundice are important. *Amyloid Liver.*—A history of suppuration or syphilis, enlargement of the spleen, urinary phenomena, in particular albumin and casts which take the iodine test, and a less rapid and marked cachexia are diagnostic. Gummatous nodules may greatly embarrass the diagnosis. *Echinococcus of the Liver.*—Ordinary echinococcus cysts are little likely to be confounded with malignant disease. The enlargement with hard nodules upon the surface, characteristic of multilocular echinococcus, may give rise to uncertainty, especially as jaundice and ascites are frequent in both conditions. The slow progress of multilocular echinococcus, the enlargement of the spleen, and the slight tendency to cachexia are important in the differentiation. Exploratory puncture may bring away softened material containing cholesterin and hæmatoidin crystals. *Chronic Interstitial Hepatitis — Hypertrophic Cirrhosis.*—The absence of emaciation and cachexia, of pain and tenderness, and of a primary focus are of diagnostic importance. The enlarged liver is less dense than in infiltrated cancer and the jaundice more variable in intensity. *Atrophic Cirrhosis.*—The muddy complexion or jaundice, wasting, ascites, and other signs of portal obstruction common to the two affections may cause great uncertainty. Differential points are an alcoholic history, tardy course, enlargement of the spleen, and the absence of primary malignant disease in other organs in cirrhosis. That

form of hepatic cancer with cirrhosis in which the liver is reduced in size cannot, in the absence of a primary focus or metastasis, be recognized during life.

Prognosis.—The outlook is in the highest degree unfavorable. The duration of the disease rarely exceeds eighteen months. Secondary cancers run a much more rapid course than the forms associated with cirrhosis. Resection of malignant growths in the liver has been performed.

IV. DISEASES OF THE PANCREAS.

i. Hemorrhage into the Pancreas.

The hemorrhage usually occurs as a manifestation of the necrosis incident to acute pancreatitis. There are instances, however, in which large hemorrhage into the organ and adjacent structures has occurred in the absence of inflammation—*Pancreatic Apoplexy.*

The etiology is that of acute hemorrhagic pancreatitis. The condition occurs in middle life.

Symptoms.—The onset is sudden. The patients are usually in their ordinary health. In some instances there have been digestive symptoms or previous attacks of biliary colic. Pain is intense and located in the upper part of the abdomen. It is sharp, sometimes colicky, and increases in severity. It is accompanied by nausea and vomiting, which are frequent and intractable but not followed by relief. The patient becomes restless, anxious, and depressed. The surface is cold and covered with a clammy sweat. The pulse is feeble, rapid, and thready. The appearance of the patient is that common in internal hemorrhage. There is epigastric tenderness followed by tympany which is usually moderate. The temperature is normal or subnormal, the patient falls into syncope which terminates fatally in the course of twenty-four or forty-eight hours.

ii. Acute Pancreatitis.

(a) ACUTE HEMORRHAGIC PANCREATITIS.

Definition.—The term acute hemorrhagic pancreatitis is employed to designate a rapidly developing destructive process,—necrosis,—accompanied by hemorrhage into the substance of the organ and adjacent parts, and in nearly all cases by disseminated areas of fat necrosis. In some instances there is no evidence of inflammation; in others there are inflammatory changes.

Etiology.—Predisposing Influences.—It is in the highest degree probable that cholelithiasis is the chief predisposing influence to acute hemorrhagic pancreatitis. This hypothesis is supported by the following facts: It has been experimentally shown that similar lesions are produced by the injection of bile into the gland by way of the duct of Wirsung; the condition is of common occurrence in individuals suffering from cholelithiasis: biliary calculi are present in the bile-ducts or in the duodenum in a large proportion of the cases: and an impacted gall-stone has been found, as in a case reported by Halstead and quoted by Opie, at the duodenal opening of the ampulla of Vater not of sufficient size to occlude

either the common duct or the pancreatic duct, and thus converting them into a continuous closed channel.

The Exciting Cause.—The statement of Opie that, "While at present it cannot be denied that other causes may produce the condition, only one etiological factor has been demonstrated, namely, the impaction of a gall-stone in the diverticulum of Vater, diverting bile into the pancreatic duct" is unquestionably true. The immediate cause then is a mechanical one. The bile and the pancreatic secretion are present at low pressure, but the bile is forced into the pancreatic duct by the contractions of the gall-bladder, and in some of the cases the walls of the pancreatic duct have been stained with bile.

In the cases in which no evidence of gall-stone disease is found upon post-mortem examination two conditions may occur: first, a neoplasm interfering with the discharge of the bile into the duodenum; and second, occlusion of a narrow canal between the ampulla of Vater and the duodenum by catarrhal swelling or a plug of tenacious mucus.

If abscess formation occurs it is due to pyogenic infection. Pus collects in the bursa omentalis and may find its way behind the peritoneum or rupture into the stomach or adjacent portion of the small intestine.

Symptoms.—No sharp line of demarcation can be drawn between acute, gangrenous, and suppurative pancreatitis, which are in fact consecutive processes in cases in which death does not take place rapidly. The attack may have been preceded by attacks of biliary colic, or it may supervene upon such an occurrence. Again, since anatomical studies of the relative diameter of the common duct and the canal leading from the diverticulum of Vater into the duodenum have shown that a small calculus readily passing through the common duct may fully occlude the duodenal opening, the onset of the symptoms of acute pancreatitis may constitute the first clinical phenomena of gall-stone disease.

There is little to add to the terse and graphic description of Fitz: "It (the attack) begins with intense pain, especially in the upper abdomen, soon followed by vomiting, which is likely to be more or less obstinate, and not infrequently by slight epigastric swelling and tenderness with obstinate constipation. A normal or subnormal temperature may be present, and symptoms of collapse precede by a few hours death, which is most likely to occur between the second and fourth days." Nausea is marked and continues between the attacks of vomiting. The vomitus is not characteristic. It does not at first contain bile. Collapse symptoms occur early and, considered in connection with the above symptoms and the rapidly fatal result, suggest acute poisoning.

Diagnosis.—Direct.—The diagnosis rests upon the sudden occurrence of the foregoing symptoms in an adult who has suffered from chronic gastroduodenal catarrh or from attacks of biliary colic; the location of the pain and tenderness in the upper abdomen; the absence of the distinct, board-like rigidity characteristic of early peritonitis and an early high leucocytosis. Da Costa, in seven counts in four cases at the German Hospital in Philadelphia, found a leucocytosis ranging from 11.000 to 30,000. If the patient survives, circumscribed epigastric fulness, which may be tense and tympanitic or dull upon percussion, may develop.

Opie has suggested that the fat-splitting ferment, which, free in the tissues, causes the fat necrosis, may be excreted by the kidneys, and, using the ethyl butyrate method of Castle and Loevenhardt, which depends upon the power of a fat-splitting ferment to decompose that substance with the liberation of butyric acid, was able in one instance to demonstrate the presence of a marked acid reaction, while a control specimen remained unchanged.[1]

Differential.—*Acute Poisoning.*—The anamnesis is important. In poisoning by meat or fish a number of persons are usually simultaneously affected. There is a period of prodromes consisting of languor, nausea, and griping pain in the belly. The attack begins suddenly with chilliness, faintness, and headache. Collapse symptoms supervene with vomiting and diarrhœa, which is often uncontrollable. In poisoning by corrosive chemicals the surrounding circumstances, certain marks upon the lips and garments, and the behavior of the patient are important. Collapse is preceded by intense pain in the stomach, followed by colic and in many instances by diarrhœa. *Strangulated Hernia.*—In a doubtful case the sites of hernial tumors are to be carefully examined; the history is important; constipation and fecal vomiting are significant. *Intestinal Obstruction.*—In acute obstruction we find constipation, abdominal pain, and vomiting. The pain is at first colicky, later continuous and severe. Vomiting is an early symptom. Nausea is less marked than retching. The vomitus consists at first of the stomach contents, then of bile-stained mucus, and finally of a darkish liquid with a fecal odor. In many cases neither fæces nor flatus are passed by the bowel; in some the contents of the bowel below the constriction are voided. Abdominal tenderness and tympany come on later. If the obstruction be seated in the small bowel the distention may be slight, but it is not confined to the epigastrium. Pain and tenderness are later symptoms and are not circumscribed. Collapse symptoms are not usually at first present. There is, as a rule, a very high leucocytosis, 60,000 or more. Acute hemorrhagic pancreatitis is very often mistaken for intestinal obstruction. *Embolism of the Larger Mesenteric Vessels—Infarction of the Bowel.*—This accident gives rise to sudden colic, nausea, vomiting, and bloody diarrhœa. The condition resembles acute obstruction, marked tympanites develops, and death occurs in collapse. *Perforative Peritonitis.*—The differentiation becomes apparent when the symptoms are enumerated. In perforation of an ulcer of the stomach, bowels, or gall-bladder, necrosis of the appendix, rupture of an abscess of the liver, spleen, kidney, or Fallopian tube, chilliness or rigor, intense abdominal pain, and exquisite tenderness are early symptoms. The pain and tenderness are general but more intense as a rule in the region of the perforating lesion. There is early spastic contraction of the abdominal muscles upon one or both sides—a very significant sign. The patient assumes and maintains an attitude by which the tension of the abdominal muscles is diminished, and lies with his head and shoulders elevated and his thighs and legs strongly flexed. Later the tension relaxes, the abdomen becomes tympanitic, and both pain and tenderness abate. When there is a history of gall-stone disease the differential diagnosis becomes as

[1] Diseases of the Pancreas, Opie, 1903, p. 322.

important as it is obscure. Absence of muscular tension, circumscribed pain and tenderness in the epigastrium, and early profound collapse are suggestive of pancreatitis.

Cammidge found that in cases of pancreatic disease a peculiar substance, probably pentose, is present in the urine and can be detected by its forming long, yellow, flexible crystals arranged in sheaves in the presence of phenylhydrazine. The test is very elaborate and can only be carried out in a laboratory.

(b) GANGRENOUS PANCREATITIS.

Etiology.—Necrosis of the whole or a portion of the gland may follow hemorrhage or hemorrhagic pancreatitis in the cases in which death does not occur in the first three or four days. The tissue of the pancreas is dry and friable, and the necrotic organ lies nearly free in the omental cavity. Death usually occurs in the course of two or three weeks. There are recorded cases in which the necrotic pancreas has been discharged by way of the rectum, with recovery.

Symptoms.—The clinical symptoms are those of acute hemorrhagic pancreatitis, but the illness is of longer duration. As the gangrenous pancreas occupies a position in the posterior wall of the lesser peritoneal cavity, peritonitis ensues, and this cavity becomes filled with pus and necrotic material. These changes are followed by fever, delirium, and the general symptoms of septic infection.

Diagnosis.—The condition cannot be positively determined during life. A probable diagnosis rests upon the occurrence of the symptoms of acute hemorrhagic pancreatitis with a prolongation of life, and the occurrence of septic phenomena.

(c) ACUTE SUPPURATIVE PANCREATITIS—PANCREATIC ABSCESS.

Suppurative inflammation of the pancreas presents nothing characteristic, with the exception that it most commonly occurs as a later stage of acute hemorrhagic and gangrenous pancreatitis. There may be a single large abscess, multiple small abscesses, or diffuse purulent infiltration. The lesser peritoneal cavity may be distended with pus.

Etiology.—Cholelithiasis constitutes a predisposing influence. The actual cause of the disease is to be found in an antecedent hemorrhagic pancreatitis. In some cases the condition has followed traumatism.

Symptoms.—The clinical manifestations follow those of acute hemorrhagic pancreatitis and are the result of the invasion of the necrotic pancreatic and peripancreatic tissues by pus-producing organisms. There may be epigastric prominence, or a deep-seated mass may be felt in the median line. Irregular chills and fever, with profuse sweating and progressive loss of flesh and strength, occur. In some instances the disease runs a protracted course with irregular fever, epigastric pain, and vomiting. Slight icterus, fatty diarrhœa, and glycosuria occur in some cases. Perforation into the stomach, duodenum, or peritoneum may occur. Portal thrombosis has been noted.

Diagnosis.—The recognition of the condition is extremely difficult. Circumscribed epigastric prominence or a resistant deep-seated mass in connection with the above symptoms is suggestive. The gravity of the condition justifies surgical diagnosis by an exploratory operation.

Prognosis.—The prognosis in the acute cases is uniformly fatal, death occurring in the course of twenty-four or thirty-six hours. In a limited number of subacute cases, in which life has been prolonged, spontaneous recovery has occurred with the discharge of a portion of the necrosed gland by the bowel, or surgical operation has been followed by cure.

iii. Chronic Pancreatitis.

The gland undergoes sclerotic changes as the result of chronic inflammation. It is sometimes diminished in size; in other cases it is larger than normal, and may form a palpable epigastric tumor. Two types of interstitial inflammation have been distinguished—an interlobar and an interacinar.

Etiology.—Predisposing Influences.—Age is important. Chronic pancreatitis is much more common between the fortieth and sixtieth years of life than at any other period. The disease is frequently secondary to disease of the intestine and bile passages, and of the liver.

Exciting Cause.—Inflammatory irritants may reach the organ by way of the duct of Wirsung or Santorini. Obstruction to the outflow of the pancreatic secretion may be followed by chronic pancreatitis. A form of interstitial pancreatitis of the new-born occurs in syphilis. A history of tuberculosis, syphilis, and the abuse of alcohol may frequently be obtained. The association of cirrhosis of the liver with chronic pancreatitis has been noted. Arteriosclerosis may cause atrophic changes.

Symptoms.—The symptoms are obscure, and the condition is rarely recognized during life. At operations for surgical diseases involving the gall-bladder and bile passages the head of the pancreas is not infrequently found enlarged and is so hard as to suggest malignant neoplasm. In such cases it has frequently happened that the patient has recovered and remained well for years. Usually there is a history of epigastric pain, nausea, and persistent vomiting. The signs of arteriosclerosis are frequently present. The enlarged head of the pancreas may press upon the common bile-duct and produce jaundice. Fatty diarrhœa, glycosuria, and diabetes may occur. The islands of Langerhans are the source of a glycolytic ferment necessary to the metabolism of the carbohydrates. Functional or organic disease of these bodies may be followed by an accumulation of glucose in the blood and by glycosuria — *pancreatic diabetes*. Minor functional derangements may give rise to alimentary glycosuria, that is, glycosuria following the ingestion at once, while fasting, of amounts of glucose less than about 200 grammes—the quantity which can be taken in health without causing glycosuria.

Hyaline Degeneration of the Pancreas.—Opie has especially drawn attention to this condition. It affects chiefly the islands of Langerhans and is associated with diabetes mellitus. It may occur as an independent condition, or may be associated with a moderate degree of increase in the interstitial tissue, or arteriosclerosis. It is equally

common in the two sexes, and has been chiefly observed after middle life. Chronic interstitial pancreatitis of the interacinous type has been present in the majority of the cases.

iv. Pancreatic Calculi.

Pancreatic lithiasis is a rare condition. It may cause chronic interstitial pancreatitis, dilatation of the duct, a large retention cyst, acute suppurative inflammation, or finally, as in the case of cholelithiasis, carcinoma.

Symptoms. — In pancreatic colic the pain is sudden, intense, and paroxysmal. It has its maximum intensity at the costal margin to the left of the middle line, and passes through to the back. Fatty diarrhœa and glycosuria occur, but are not constant. Vomiting may occur. Pancreatic calculi are in some instances associated with gall-stone disease.

Diagnosis. — Direct. — The occurrence of the above symptoms in paroxysms, usually at intervals of months or years, without jaundice, is suggestive. The presence in the stools, after the attack, of round, smooth or rough, opaque, white calculi, which are composed chiefly of calcium carbonate, renders the diagnosis positive.

Differential.—The symptoms when small stones are passed may suggest gastralgia or gastric ulcer; in other cases cholecystitis or a biliary calculus in the cystic duct. The focus of pain upon the left side and the character of the stones, if passed, are diagnostic.

v. Pancreatic Cysts.

The term pancreatic cyst has been used to designate any cystic tumor in, or associated with, the pancreas, although such tumors differ among themselves, etiologically, in situation and in clinical features.

Varieties.—Congenital cystic disease; retention cysts; proliferative cysts; hemorrhagic cysts; hydatid cysts; and pseudocysts.

Etiology.—Sex is without influence, the condition having been observed in about the same number of men and women. Age is important, the largest proportion of cases reported being between twenty and forty years. Cases have been noted in the new-born, and between the sixtieth and seventieth years. The greater number of cysts are caused by traumatism, inflammation, or impacted calculi. They may occupy any part of the gland. Congenital cystic disease may occur in the pancreas as in the kidney and liver. The causes of retention cysts are the impaction of calculi, cicatricial stenosis, pressure upon the duct, and dislocation of a part of the organ. Proliferative cysts are of two kinds: simple, or cyst adenoma, and malignant, or cystic epithelioma. There is a marked tendency to hemorrhage into pancreatic cysts. Collections of blood in the substance of the organ are characteristic of the acute forms of necrosis and inflammation. Hydatid cysts are exceedingly rare. Pseudocysts are circumscribed collections of fluid found in the proximity of the pancreas, but not having their origin in the substance of the gland. They commonly occupy the lesser peritoneum.

The fluid is usually thick and viscid, alkaline, of variable color, clear, milky, yellow, green, or brownish-black. There is usually, in the colored fluids, an admixture of blood. The specific gravity varies from 1.010 to 1.020 or higher. Serum albumin, paraglobulin, mucin, and urea are present. The presence of fat-splitting, proteolytic, or diastasic ferments cannot usually be demonstrated, although the last may sometimes be found. The secretion of the chemically inflamed pancreas may contain only traces of these ferments, and cannot find its way into the cyst unless there is free communication with the glandular parenchyma. Moreover, similar ferments have been demonstrated in the contents of mesenteric and ovarian cysts. The discharge from a fistula resulting from operation may contain the pancreatic ferments.

Symptoms and Signs.—In small cysts symptoms are absent or indefinite. In larger cysts there are pressure symptoms, especially epigastric discomfort, weight, and fulness. Pain, especially after food, confined to the upper part of the abdomen, radiating to the back and toward the left, vomiting, and constipation occur. Jaundice, usually slight, may result from the pressure of a cyst in the head of the pancreas upon the common duct. Light-colored stools containing free fat and much undigested muscle fibre are significant but by no means constant. Alimentary glycosuria and diabetes occur only in cases in which there is extensive destruction of the gland.

The cystic tumor lies behind the posterior layer of peritoneum, which forms the lesser sac. In the great majority of cases the enlarging tumor displaces the stomach upward and to the right, and the transverse colon downward, and approaches the surface below the greater curvature of the stomach; occasionally it presents above the upper border of the stomach; and finally it may push itself between the layers of the transverse mesocolon and force the transverse colon before it, or displace both the transverse colon and the stomach upward.

The tumor occupies the epigastrium, at first usually to the left of the middle line between the costal border and the umbilicus. It may lie in the middle line, or more rarely to the right. It is usually smooth, spherical or oval, elastic, and tense. It may attain enormous dimensions and reach to the symphysis pubis, suggesting an ovarian or parovarian cyst. It is commonly immovable, or but slightly movable, either upon pressure or with the respiration, but in rare cases may be feebly so in consequence of its attachment to the tail or body of the pancreas by a narrow pedicle. When small it may resemble a solid mass, and transmitted aortic pulsation may suggest an aneurism. In large pancreatic cysts fluctuation can almost always be elicited. There is flatness upon percussion.

Diagnosis. — Direct. — The presence of a cystic tumor located—or at first located—in the upper abdomen and having the above features is of diagnostic significance. Inflation of the stomach and colon may be important, especially in small cysts. The contents may be removed for examination by aspiration—an unsafe procedure, to be employed only in extreme cases. A history of recent traumatism, as a kick or blow upon the epigastrium, is important.

Differential.—Various cystic tumors present points of resemblance to pancreatic cysts. Among the more important are the following: *Ovarian Cysts.*—Only enormous cysts of the pancreas can give rise to difficulties. Examination of the pelvic organs, preferably in the Trendelenburg position, the distention of the colon with air, and the history of the case will remove every doubt even in those cases in which the appearance of the abdomen closely resembles that caused by tumor of the ovary. *Cysts of the liver* are usually hydatid. When in the left lobe, they can scarcely be differentiated from pancreatic cyst except by aspiration of the fluid or an exploratory operation. An *enormously distended gall-bladder* may simulate pancreatic cyst. *Cyst of the suprarenal capsule,* especially upon the left side, may be readily mistaken for cyst of the pancreas. The differential diagnosis by physical signs alone is not possible. *Cysts of the Kidney, Hydronephrosis, Pyonephrosis.*—The tumor moves to some degree with respiration; it is distinctly unilateral, and occupies the lumbar region rather than the epigastrium. The relation of the colon to it may be determined by percussion after inflation. Renal symptoms, such as aching pain, frequent micturition, and the previous disappearance of the tumor with the passage of a great quantity of water, or hemorrhage from the genito-urinary tract, point to the kidney. *Cysts of the Mesentery.*—These tumors are characterized by their location near the umbilicus, movability in a lateral or rotary direction, and tympany around the circumference of the mass, and in a band across it. If a pancreatic cyst develops between the layers of the transverse mesocolon the band of resonance will be due to the colon. *Omental Cysts.*—A cyst developing in the omentum directly below the stomach would push the stomach up and the transverse colon down. By the physical signs the diagnosis would not be possible. The anamnesis and the general symptoms might justify a provisional diagnosis. *Cysts of the posterior wall of the stomach, cysts of the spleen, and certain retroperitoneal cysts* are of very rare occurrence and present unusual diagnostic difficulties. In many of the cases the differential diagnosis between these tumors and cysts of the pancreas is impossible.

Prognosis.—The condition, if early diagnosticated and relieved by operation, usually terminates in recovery. To this statement a reservation must be made in regard to hemorrhagic cysts.

vi. Tumors of the Pancreas.

Neoplasms are rare. Sarcoma, adenoma, and lymphoma are extremely infrequent. Carcinoma mostly involves the head of the organ, is often primary, and commonly occurs after middle life. Miliary tubercle is an accompaniment of acute miliary tuberculosis. Syphilis occurs in the form of chronic interstitial inflammation or gummata. The frequency with which the head of the organ is involved, especially in carcinoma, accounts for certain symptoms. The tumor exerts pressure upon the common bile-duct, the pancreatic duct, the duodenum, and the pylorus. The stomach, colon, aorta, vena cava, portal vein, superior mesenteric vein, and the splenic artery and vein may also be compressed. The breaking down of the growth may cause perforation of any of these organs.

Etiology. — Carcinoma. — Men are more frequently affected than women. The condition has been found with greatest frequency between forty and sixty years.

Symptoms. — In some cases the symptoms are obscure. Generally epigastric pain radiating to the back, often intense and aggravated at night, is a prominent symptom. Jaundice, a gall-bladder tumor, and enlargement of the liver are common. A tumor in the pyloric region, and fixed, may often be made out. There is rapid wasting. Constipation is usual; the stools are large and contain free fat and undigested muscle fibre in unusual amount. Sugar may be present in the urine, and the symptoms of diabetes may be noted. A subnormal temperature also occurs. A dense shadow may be noted upon X-ray examination.

Diagnosis.—Direct.—The occurrence of the above symptoms with cachexia renders the diagnosis of carcinoma of the pancreas positive.

Differential. — In default of a definite symptom-complex the possibility of interstitial pancreatitis, or malignant disease of the common duct, the liver, or the pylorus is to be considered. *Chronic Pancreatitis.*—A long history, recurrent painful attacks, and epigastric tenderness are in favor of benign disease; loss of flesh is less marked than in carcinoma, and vascular disturbances and dropsy are far less common. Anæmia is less pronounced, and cachexia is absent. *Cancer of the Bile-ducts.*—There is almost always a history of gall-stone disease. If the duct of Wirsung be involved the differential diagnosis cannot be made; if not, the destructive signs of pancreatic disease, especially the rapid wasting, will be absent. *Cancer of the Liver.*—Enlargement of the liver with nodules upon its surface and borders, and moderate jaundice, or its absence, are diagnostic. *Cancer of the Pylorus.*—Marked gastric symptoms, retention vomiting, dilatation of the stomach, absence of free hydrochloric acid, and the presence of altered blood in the vomited matters are against pancreatic carcinoma alone, but the conditions are frequently associated.

V. DISEASES OF THE PERITONEUM.

i. Ascites.

Abdominal Dropsy—Hydroperitoneum.

Definition.—An accumulation of serous fluid in the peritoneal cavity.

Etiology.—General Causes.—The accumulation may be part of a general dropsy caused by derangement of the mechanism of the circulation, as in disease of the heart. In some cases of heart disease the dropsical effusion may be limited to the peritoneum. Ascites occurs also in various forms of hydræmia and in advanced renal dropsy. Local Causes.—Chronic inflammation of the peritoneum; portal obstruction; abdominal tumors.

Character of the Ascitic Fluid.—The fluid in ascites from stasis is clear, yellowish or greenish-yellow, alkaline in reaction, of a specific gravity of 1.010–1.015, and contains in solution the soluble substances of the blood. Red blood-corpuscles are also present, usually in small numbers.

In *inflammatory ascites* the fluid contains flakes of fibrin, other masses of coagulated material, large numbers of pus-corpuscles when it is purulent, and many blood-corpuscles when it is hemorrhagic. In cancerous ascites molecular débris and cancer-cells may be present. Various bacteria, as streptococci, staphylococci, colon bacilli, gonococci, B. typhosi, pneumococci, and tubercle bacilli may also be present. The presence of tubercle bacilli may be determined by inoscopy. The differential diagnosis between transudation and exudation cannot always be made from the characters of the fluid alone. For this purpose neither the albumin percentage nor the specific gravity avails. Cytodiagnosis is far less useful in the determination of the nature of peritoneal than that of pleural effusions. The effusion in cirrhosis is sometimes darker in color than in other conditions, and that in cancer and tuberculosis is usually hemorrhagic. In the cells of carcinomatous ascites mitosis is more common than in other forms, and the cells in melanotic sarcoma may contain pigment granules, though the fluid is clear.

Chylous and Chyliform Ascites.—The fluid is milky in appearance and resembles chyle. Quincke recognized two essentially different forms, one a transudate, — *ascites chylosus*, which owes its characters to the actual presence of chyle, — the other *ascites adiposus*, — chyliform or pseudochylous, — the appearance of which is due to the admixture of fat derived from the metamorphosis of the disintegrating cells of an inflammatory exudate or the endothelium. Fat is present in both forms in minute dust-like particles. In chylous ascites there are very few cells in a state of fatty degeneration. In chyliform ascites, on the other hand, there are many cells containing fat granules. The presence of sugar does not justify the conclusion that a milky ascites is chylous, since it has been definitely established that sugar may be present in any form of transudate or exudate in the serous sacs. Both of these fluids, subjected to agitation with ether after the addition of potassium hydroxide, clear up to a greater or less extent, and both respond to the osmic acid and other tests for fat. They are bacteria free and do not undergo decomposition for indefinite periods. Upon standing they separate into a thick sedimentary layer, and a fatty, cream-like layer at the top.

Milky, Non-fatty Ascites.—The fluid resembles the fatty forms, but neither microscopically nor clinically reveals the presence of fat. The milk-like appearance has been ascribed to various proteid and mucoid substances, and to lecithin, but the subject is still under investigation.

Hæmoperitoneum may result from traumatism and the rupture of viscera, as the liver, spleen, or mesentery. It occurs also in extra-uterine fetation and the rupture of aneurism. Recurrent hemorrhage into the peritoneum has been observed in the absence of assignable cause. Blood-stained fluid may be present in acute pancreatitis, volvulus, twisting of the pedicle of an ovarian cyst, and other similar conditions.

Symptoms.—These vary according to the amount of fluid. Small amounts occasion no discomfort, and the gradual accumulations of a considerable ascites may not be realized by the patient. It is very common in the ascites associated with cirrhosis of the liver for the patient to first become aware of his condition by his inability to make his waistband

meet. Large accumulations of fluid give rise to much distress by pressure and tension of the abdominal walls, sensations of fulness and weight, and interference with the play of the diaphragm, causing dyspnœa and cyanosis. Œdema of the lower extremities and pudenda, occurring subsequently to large ascites, is due to interference with the return of the blood by the pressure of the fluid upon the ascending vena cava, and interference with the action of the heart by upward pressure against the diaphragm. The superficial venous trunks—mammary, epigastric—are frequently widened and tortuous, and reveal, upon stripping, the upward current of the contained blood.

Physical Signs.—(See Methods of Physical Diagnosis, Part II, Vol. I.)

Diagnosis.—Small quantities of fluid gravitate into the pelvis and fail to manifest themselves by the signs elicited upon external examination. If necessary, the patient may be placed in the knee-elbow posture when a small collection of ascitic fluid gravitating into the most dependent region may be recognized upon percussion, or a finger may be lightly pressed into the inguinal ring, the patient being in the erect posture. Gentle tapping will cause a wave which is felt by the finger. Again the finger, introduced into the rectum or vagina, the patient being in such a position that the fluid gravitates into the cul-de-sac of Douglas, may perceive the fluctuation produced by tapping on the lower part of the belly wall. Less than 1500–2000 c.c. cannot be detected by physical examination.

Paracentesis Abdominis.—The patient should sit on a chair or upon the side of a low bed, with his knees separated and a large jar or small tub between his feet. The puncture should be made in the median line midway between the symphysis and navel, under strict antiseptic precautions, with a straight trocar, one-eighth of an inch in diameter; it should be determined by previous percussion that an intestinal coil does not lie beneath the point at which the patient is to be tapped. Pressure should be applied by a many-tailed bandage to the abdominal wall as the fluid escapes. If the canula becomes obstructed by floating intestine, a change in direction or the insertion of a probe will clear it. Fluids may be allowed to escape until the flow ceases. A strip of adhesive plaster may be placed over the opening, some oozing from which is likely to occur.

Differential Diagnosis.—*Large cysts* may simulate ascites. The error is most common in *cysts of the ovary;* much more rare are pancreatic cysts of such size. There is tympanitic resonance in the flanks, and the circular or oval area of tympany is not present in the umbilical region. Examination per vaginam may yield important information. *Enormous lipomata* have been mistaken for ascites. There is an obscure sense of fluctuation, but the shifting areas of tympany characteristic of ascites do not occur. The condition is exceedingly rare; females are chiefly affected; the tumor develops in middle life, and is of slow growth. An *overdistended bladder* may reach to the umbilicus, or above it, and has been mistaken for ascites. The dribbling of urine,—incontinence of retention,—and the outline of the swelling should put the practitioner upon his guard. The catheter will at once settle the matter. *Encysted inflammatory exudates*, when large, may simulate ascites, especially when pain and fever,

or other constitutional symptoms, are no longer present. In most cases the careful employment of the methods of physical examination is adequate for the diagnosis.

ii. Acute General Peritonitis.

Definition.—Acute diffuse inflammation of the peritoneum.

Etiology.—The peritoneum is peculiarly exposed to local and general infection by traumatism, extension from the viscera which it invests, the perforation of hollow viscera, rupture of the capsules of organs or the walls of abscesses, and by way of the lymph channels and the blood. Peritonitis may be primary or secondary.

1. Primary Peritonitis.—Infection takes place by the blood or by the lymph stream, and not as the result of any lesion of the viscera with which the peritoneum is in relation, or any wound or surgical operation. Acute general peritonitis is sometimes attributed to exposure to cold or damp, and has then been described as rheumatic. The form which occurs as a terminal event in renal disease, gout, and arteriosclerosis is more common. Whether or not these forms of peritonitis—idiopathic peritonitis—are in the strict sense primary remains unsettled.

2. Secondary Peritonitis.—From the point of view of the source of infection, three groups of cases may be recognized. (a) Those in which the infection of the peritoneum takes place from without by way of traumatism or surgical operation. (b) Those in which one of the abdominal organs, or an abscess, ruptures and its contents are discharged into the peritoneal cavity. Intestinal perforation is the most common accident of this kind. (c) The cases in which bacteria find their way through the wall of the intestine in the absence of a large or small solution of continuity, or enter the peritoneal cavity by way of the lymph channels, as in certain forms of puerperal peritonitis, or peritonitis consecutive to infection of the pleura.

Bacteriology of Acute Peritonitis. — One or several varieties of bacteria may be found in the exudate—single or mixed infection. Those which are the most important are the Streptococcus pyogenes, the Diplococcus pneumoniæ, and the Bacillus coli communis. These are frequently present alone — monoinfection. In rare instances the Staphylococcus pyogenes aureus has been found in pure culture in the peritoneal exudate. The gonococcus may also cause peritonitis, whether in simple or mixed infection has not yet been positively determined. In simple infection the following organisms have also been found: Micrococcus lanceolatus, B. pyocyaneus, and the B. influenzæ. In so-called primary—idiopathic—peritonitis, and in postoperative peritionitis, simple infection is much more common than mixed infection; in the secondary forms mixed infection is more common, but monoinfection may occur.

Clinical Etiology.—The vast majority of cases which arise in consequence of disease of the abdominal or pelvic organs invested by peritoneum, or in near topical relation to the peritoneal cavity, are demonstrably "secondary." The organs most commonly involved in the primary affection are the intestines and the reproductive organs in the female.

Intestines.—In the order of frequency and importance, lesions of the vermiform appendix stand first. Intestinal ulceration, especially the ulcers of enteric fever and peptic ulcer in the duodenum, come next. Other forms of intestinal ulcer, as tuberculous, dysenteric, diphtheritic, are less apt to cause perforation, and when this accident occurs it is usually into a region of the peritoneum shut off by adhesions and often already the seat of a circumscribed abscess. Certain forms of intestinal ulcer, both acute, as after extensive burns of the surface, or peptic ulcer, and chronic, as those which occur occasionally in scurvy and leukæmia, show no tendency to perforation. Carcinoma causes chronic adhesive peritonitis and circumscribed abscess formation, but scarcely ever acute diffuse inflammation of the peritoneum. *Acute Occlusion of the Bowel.*—Volvulus and strangulation and less frequently intussusception give rise to peritonitis. Chronic stenosis is rarely the cause of this condition. Rupture of the bowel, like perforation, is at once followed by general peritonitis.

The Stomach.—Peptic ulcer and carcinoma may prove the point of departure for acute peritonitis. Sudden perforation, before adhesions have formed, may occur; more commonly, adhesions with adjacent viscera take place as the result of a local peritonitis, with abscess formation or perforation into the colon.

Liver, Gall-bladder and Bile Passages.—Perihepatitis and local adhesive inflammation are common in diseases of these organs, but acute diffuse peritonitis is infrequent. It may result from rupture of an abscess or hydatid cyst, or from strangulation or acute intestinal obstruction caused by pericholecystitic adhesions. An abscess about an infected gall-bladder has, in rare instances, ruptured into the general cavity of the peritoneum.

The Spleen.—Acute diffuse peritonitis arising from disease of this organ is rare. Rupture of the capsule from traumatism, and the rupture of an abscess before adhesions to neighboring viscera have formed, are the two principal events.

The Pancreas.—Acute hemorrhagic pancreatitis gives rise to inflammation of the lesser peritoneum, and when the patient survives for a period general peritonitis may occur by extension. The rupture of a pancreatic abscess into the general peritoneum is an extremely rare event.

Kidneys and Bladder.—Acute diffuse purulent peritonitis occasionally results from the rupture of an abscess of the kidney. Rupture of a hydronephrosis may not be followed by peritonitis, if the urine does not contain pathogenic bacteria. Perforation of the bladder as the result of ulceration, or the more serious forms of diphtheritic ulceration, may result in a localized abscess or in acute peritonitis.

The Genital Organs.—Acute gonococcus peritonitis, having its starting-point from the vas deferens or seminal vesicles, is exceedingly rare. In the female, however, the sexual organs constitute the most frequent starting-point of acute peritonitis. Puerperal peritonitis is the most common form. Gonococcus infection is an extremely common cause of pelvic peritonitis, and occasionally, in young girls, of acute general peritonitis, which may also occur in children suffering from vulvovaginitis by extension to the tubes.

Lesions of the Abdominal Parietes, Mesenteric and Retro-peritoneal Glands, and Inflammatory Disease of the Other Serous Cavities.—Peritonitis sometimes arises in consequence of inflammation or suppuration in the abdominal walls, the burrowing of psoas or other abscesses, rectal disease, or caries of the vertebra, ribs, or bones of the pelvis. It may also be secondary to disease of the mesenteric glands, especially in tuberculous disease, and enteric fever—pseudo-abscess. Pleurisy, pulmonary abscess, gangrene of the lung, and purulent pericarditis may be followed by acute general peritonitis in consequence of infection through the diaphragm.

Acute Infectious Diseases.—Acute peritonitis sometimes occurs in the course of rheumatic fever. It is common in septic conditions and especially in puerperal sepsis. It is extremely rare in the continued and eruptive fevers, and when it occurs in the course of these infections is secondary to some local lesion, as perforation in enteric fever. It sometimes occurs in erysipelas, especially when the abdominal walls are involved, in influenza of the gastro-intestinal form, and in pneumonia.

Peritonitis in the Fœtus and New-born.—A septic peritonitis of the fœtus arises in consequence of infection from the mother by way of the placental circulation. Not alone the peritoneum, but also the other serous cavities, the pleuræ, pericardium, and meninges are affected. In the new-born, infection by way of the umbilical wound, usually through the lymph channels, is a frequent cause of general peritonitis.

The blood-vessels of the peritoneum in recent cases are more or less deeply injected, and the coils of intestine distended and bound together by lymph. The exudate may be fibrinous, serofibrinous, purulent, gangrenous, or hemorrhagic. The fluid exudate varies in amount from a few small collections of clear serum among the adherent loops of bowel to many litres.

Symptoms.—The symptoms of peritonitis in general may be arranged in the following groups: (a) Symptoms immediately due to the peritoneal inflammation,—pain and the phenomena denoting the presence and amount of the exudate. (b) Symptoms caused by derangements of organs and structures implicated in the process, as the stomach, intestine, bladder, abdominal muscles, and diaphragm,—vomiting, constipation, meteorism, frequent and painful micturition, early rigidity, and late paresis of the abdominal walls, hiccough. (c) Constitutional or toxæmic symptoms: fever with its attendant phenomena, circulatory disturbances, anæmia, modifications of the urine, nutritional disorders, and manifestations of sepsis. These symptoms show great variation in their intensity and association in the different forms of peritonitis and in different cases.

(a) **Peritoneal Symptoms.**—Pain and tenderness in the abdomen characterize the onset. The pain is severe and continuous, the tenderness exquisite. In non-perforative cases the pain gradually reaches its maximum; in perforative cases it is almost always extremely severe from the onset. In enteric fever and other stuporous states the occasional absence of pain is due to the mental condition. The pain is continuous, and not only is it increased by pressure, but also by movement. The patient lies motionless in the dorsal posture, with the legs and thighs flexed. Respiration is shallow, rapid, and of the costal type. Cough is suppressed, and sneezing

is attended with agonizing pain. To this persistent, characteristic pain is superadded colic due to intestinal peristalsis, and recurring in paroxysms. The pain and tenderness may be present uniformly over the whole abdomen. It is very often most intense below the umbilicus. Frequently, but not invariably, these symptoms are most intense in the area corresponding to the starting-point of the inflammation, as the ileocæcal region, the pelvis, or the epigastrium.

A fibrinous exudate sometimes manifests itself by a friction sound, usually best heard in the upper part of the abdomen, over the liver or spleen. A fluid exudate gradually collects in a majority of the cases. Its presence may be first recognized by dulness in the flanks. As it increases, it gives rise to the characteristic physical signs described under the heading "Physical Diagnosis" (see Part II, Vol. I).

(b) **Visceral Symptoms.**—Vomiting is one of the earliest symptoms in acute peritonitis, and greatly increases the pain. It usually continues several days, not ceasing until the fatal termination, an improvement in the patient's condition, or the outpouring of a large exudate. In the last instance, the cessation of vomiting may be an unfavorable sign. The vomitus consists, at first, of the gastric contents; later, of a bile-stained greenish fluid, and in some instances of blackish material with a fecal odor. Vomiting may be absent in large perforation of the stomach. Constipation is the rule; in some cases the bowels move spontaneously every day or two; in puerperal septic peritonitis diarrhœa is common. Hiccough is a common and distressing symptom.

Muscular rigidity, the result of reflex irritation, is an early and extremely valuable sign of acute peritonitis. It may cause retraction, even a scaphoid abdomen, and by restraining peristalsis may diminish the pain. It is especially marked in peritonitis due to perforation. In rapidly fatal cases the abdomen may be flat and rigid throughout the entire course of the attack.

Painful micturition is due to traction exerted upon the inflamed peritoneum by the contractions of the bladder. Retention of urine is common, especially in men. More often there is great vesical irritability, with frequent micturition. The urine is diminished, high colored, and often contains albumin. It is characteristic of acute diffuse peritonitis that the urine contains large quantities of indican. The micro-organisms which cause the disease have been present. As the rigidity passes away, meteorism takes its place. It is due to paresis of the intestine, and may appear early in the disease, especially in the perforative cases. In extreme cases the bowel is completely paralyzed, and no auscultatory signs of peristalsis can be heard, the belly is enormously distended, especially in its upper and middle segments, and the skin is tense, smooth, and glistening. In puerperal peritonitis the distention, owing to the relaxation of the stretched walls, is greater than in other forms. The splenic dulness may be obliterated; the liver dulness is greatly diminished and may wholly disappear in the midclavicular line; the diaphragm is pushed up so that the apex beat of the heart may be felt in the fourth intercostal space. The obliteration of liver dulness in the front of the body may be due to tympany alone, and is not, therefore, a positive sign of pneumo-

peritoneum or of the perforation of an air-containing viscus. If, when the patient is turned upon his left side, dulness disappears in the axillary line, there is free air in the cavity of the peritoneum.

(c) **General Symptoms.**—The attack begins, in a majority of the cases, with chilliness or a rigor. Fever follows, but is not constant and does not conform to type. The temperature may rise suddenly to a considerable height, but does not often exceed 104° F. (40° C.), or it may gradually rise for several days. In either case it becomes irregular or drops to normal as the attack progresses. As death approaches, the temperature may show rapid oscillations. In perforative peritonitis, the temperature very often drops to subnormal ranges,—temperature of collapse,—and remains there until death. The pulse is rapid, small, and wiry. Its frequency is 120–160 per minute and bears no constant relation to the temperature.

Leucocytosis of the polynuclear neutrophilic type, 18,000–40,000, is found, except in the gravest cases, in which there may be leucopenia.

The clinical picture of acute diffuse peritonitis, from the time that the disease is fully established, is very characteristic. The facies indicates great suffering and anxiety and presents the signs of collapse. The nose is pinched and pointed, the eyes are sunken, the temples flattened; there is cyanosis, and the brow is wet with drops of sweat—*facies Hippocratica.* The patient lies motionless, the respirations are shallow and rapid, the pulse is thready, the knees are drawn up, the hands and feet cold and shrunken.

This form of peritonitis usually terminates fatally. The perforative forms often run their course within forty-eight hours; the non-perforative forms in four or five, or sometimes in eight or ten, days. Exceptionally death occurs very suddenly, with signs of cardiac paralysis.

Diagnosis.—Direct.—The diagnosis rests upon the sudden onset of intense abdominal pain, tenderness, fever, vomiting, rigidity of the abdominal muscles, and collapse symptoms. When the attack is fully developed the facies and attitude are very suggestive. Inquiry into the previous health will often reveal the primary cause of the attack. It is to be borne in mind that intestinal perforation and disease of the pelvic organs in females are the most common primary conditions. A history of attacks of pain in the iliac region suggests perforating appendicitis; of pain after eating, epigastric tenderness, hæmatemesis, or dark blood in the stools, peptic ulcer; of recent headache, nose-bleeding, prostration, and diarrhœa, the ambulant form of enteric fever. In females recent abortion or confinement, acute suppurative disease of the pelvic viscera, or salpingitis are common antecedent conditions. In enteric fever the signs of perforation may be masked by the patient's mental condition. In many of the cases the previous condition cannot be determined.

Differential.—The following conditions are often mistaken for peritonitis: *Acute Enterocolitis.*—The pain is colicky, and less continuous; the tenderness is less acute, and more limited; diarrhœa is a more prominent symptom, and early rigidity and subsequent tympany are not so conspicuous. In the severe cases there may be a very marked degree of collapse. *Intestinal obstruction, volvulus, and strangulation* may not only cause peritonitis, but they often also simulate it, the symptoms in common

being pain, tenderness, vomiting, and tympanites. Muscular rigidity is not so marked, local distention of the bowel, violent peristaltic movements, and the more tardy development of tympany and collapse point to occlusion of the bowel rather than inflammation of the peritoneum. *Rupture of a tubal pregnancy or an abdominal aneurism* may give rise to symptoms suggestive of perforative peritonitis. The history is very important. Restlessness and air hunger are much more marked in large internal hemorrhage than in inflammation. Embolism of the superior mesenteric artery may be attended with sudden agonizing pain, frequent vomiting, collapse, and tympany. *Acute Hemorrhagic Pancreatitis.*—A history of gall-stone disease and the localization of pain in the epigastrium are important in the diagnosis. In perforative and rupture cases, in which the peritoneum is suddenly flooded with the contents of the intestine or pus, death frequently takes place from shock in the course of a few hours, before an actual inflammation has time to develop. *Hysteria* may mimic peritonitis.

iii. Acute Circumscribed Peritonitis.

This form of inflammation of the peritoneum is, (1) adhesive, or (2) purulent.

1. Adhesive inflammation is of very frequent occurrence in local disease of the abdominal organs. It is usually narrowly circumscribed, and unattended by other immediate symptoms than pain and tenderness, the manifestations of the primary affection dominating the clinical picture. The anatomical changes consist in vascular injection, fibrin formation, and slight serous exudation. The organs involved are chiefly the liver, gall-bladder and bile passages, spleen, stomach, coils of intestines, the appendix vermiformis, and the sexual organs in the female. Perihepatitis, perisplenitis, either circumscribed or involving the whole organ, belong to this category. Intestinal adhesions following local disease or operation, adhesive inflammation affecting the gall-bladder, bile-ducts, duodenum, and the pyloric end of the stomach, or a similar process involving the Fallopian tubes or ovaries may be the cause of distressing subsequent symptoms. Tuberculous, cancerous, suppurative, or hydatid disease may be the cause of localized peritonitis.

2. Purulent.—Infection with pus-producing micro-organisms may be primary and cause acute suppurative circumscribed peritonitis, or it may occur later and lead to the formation of localized abscess and small pockets of pus among the adhesions and other lesions resulting from the non-suppurative form, so that a transitional condition may be recognized. There are certain points in which acute suppurative circumscribed peritonitis preferably arises; among these, the region of the appendix, the pelvic organs in the female, and the lesser peritoneum are most important.

(a) *Appendicular Abscess.*—The most common cause of acute circumscribed suppurative peritonitis is appendicitis—a condition fully described under the heading "Diseases of the Intestines" (q. v.).

(b) *Pelvic Peritonitis.*—Suppurative inflammations, septic, tuberculous, or gonorrhœal, are very common. They result in the formation of perimetric and parametric abscesses. Salpingitis and abscesses of the

broad ligament occur. Suppuration is frequently preceded by extensive adhesive inflammation. General peritonitis may arise by extension of the infection, or by rupture.

(c) *Subphrenic Peritonitis.* — Inflammation may involve the lesser peritoneum alone, and inflammatory exudates may be confined to its cavity. Perforating ulcers of the stomach, duodenum, or colon are sometimes so situated that they communicate directly with it, and into it pancreatic hemorrhages and abscesses may be discharged. Effusions into this space may cause an oval, smooth, tense tumor, extending into the epigastric, umbilical, and left hypochondriac regions, and simulating a pancreatic cyst. The physical signs vary greatly from time to time, according to the condition of the adjacent stomach. If the latter is distended with food, the line of demarcation between it and the tumor cannot be made out either by percussion or palpation, while if it is filled with gas, it may yield tympanitic resonance over the greater part or the whole of the tumor, causing it at times to altogether disappear. A subphrenic abscess has, in rare cases, followed pneumonia or empyema; more frequently it results from an appendicular abscess, a renal or hepatic abscess, or trauma. It may occur in connection with cancer of the stomach.

The diagnosis of simple subphrenic abscess is difficult, because the signs and symptoms are very frequently indefinite. The subjective symptoms attract attention to the upper part of the abdomen. Among these pain is most important, and may be referred to the right or left side, the back, and so forth, according to the seat of the abscess. It may be localized, or radiate into the abdomen or lower thoracic belt. When the pain is local it is usually associated with tenderness. Circumscribed phenomena in the epigastrium or left hypochondrium are suggestive. Fluctuation is rare and present only when the abscess is superficial. In rare cases there is circumscribed œdema of the overlying skin.

Pyopneumothorax Subphrenicus — *Leyden.* — When the subphrenic abscess is due to a perforating peptic ulcer air is also almost always present and the condition simulates pneumothorax.

Symptoms. — The nature of the condition is obscure and in a majority of the cases not recognized *intra vitam.* The symptoms vary according to the cause. The pus collection between the liver and the diaphragm, whether in relation with the right or the left lobe when air is not also present, closely simulates an encysted empyema at the base of the pleural sac. When it occurs in association with pneumonia, or empyema, or an abscess in a neighboring organ, the symptoms of the primary affection are more or less rapidly, often suddenly, reinforced by those of the new affection, namely, severe epigastric pain, urgent and persistent vomiting, and respiratory embarrassment. In rapidly developing cases shock may also occur. Later symptoms are chills, irregular fever of septic type, anæmia, and rapid wasting. Burrowing may occur into the pleura or, in the case of pleural adhesions, into the lung, with paroxysmal cough and copious purulent expectoration. When caused by a perforating ulcer of the stomach or duodenum, the onset is abrupt, with great pain, and the vomited material is bilious or bloody. When the abscess cavity contains gas, the diaphragm may be forced upward upon the right side as

far as the third rib, and the liver displaced downward; when upon the left side, the heart is displaced upward.

Diagnosis. — Subphrenic pyopneumothorax is very frequently overlooked. The physical signs are those of pneumothorax or pyopneumothorax upon the right or left side, according as the abscess cavity is situated upon the right or the left side of the suspensory ligament. The antecedent symptoms, in the majority of the cases, point to disease of the abdominal organs and not to disease of the lungs or pleuræ. Upon forced inspiration the lower border of the compressed lung is depressed in subphrenic abscess; the liver is usually depressed to a remarkable degree, and its lower border is distinctly palpable. The heart is displaced upward rather than laterally. In both conditions the intercostal spaces may be either obliterated or bulging. As the greater number of cases are the result of perforating peptic ulcer, the local symptoms appear very suddenly, while the general symptoms are usually more severe than in ordinary cases of pneumothorax. Exploratory puncture may be made for diagnostic purposes. The presence of material from the gastro-intestinal tract at once determines the differential diagnosis. The position of the diaphragm may be positively determined by skiagraphy. In pyopneumothorax it forms the floor, in pyothorax subphrenicus the roof, of the abscess cavity.

iv. Chronic Peritonitis.

(a) **Local Adhesive Peritonitis.**—The inflammation of the peritoneum which follows operations or accompanies local disease of the abdominal viscera is more frequently chronic than acute. When it involves coils of intestines, it gives rise to partial stenosis with constipation and colicky pains, and may ultimately be the cause of acute obstruction of the intestine by strangulation.

(b) **Diffuse Adhesive Peritonitis.**—In tuberculosis and general carcinomatous infiltration of the peritoneum the adhesions are sometimes so extensive as to entirely obliterate the cavity. This form of peritonitis is rare in other conditions, but has been encountered in tumors of the peritoneum, and after trauma. It occurs also in syphilis during intra-uterine life.

Symptoms.—The condition may not be attended with definite symptoms. Pain and tenderness are usually present.

(c) **Chronic Proliferative Peritonitis.**—There is great thickening of the membrane, without extensive adhesions. Moderate serous effusion may be present. The mesentery is shortened, and the omentum may be rolled into a firm transverse tumor. In some instances there is a general chronic inflammation of the serous membranes,—Concato's disease, polyorrhomenitis,—involving with the peritoneum both pleuræ and the pericardium. This form of peritonitis occurs in the subjects of chronic alcoholism, in chronic passive congestion, and in tumors, but is especially associated with cirrhosis of the liver.

Symptoms. — The disease may be latent, the symptoms being subordinated to those of the primary condition. They comprise abdominal uneasiness and distention, colicky pains, constipation, and diarrhœa. Jaundice is sometimes present. Ascites may occur, or the shortening of

the mesentery, the consequent drawing together of the intestines into a tumor-like mass, and the retracted and indurated omentum may simulate tumors of various abdominal organs.

Diagnosis. — The DIRECT DIAGNOSIS rests upon the concurrence of alcoholism, cirrhosis of the liver, chronic intestinal disease, chronic nephritis, with symptoms of peritoneal disease, and ill-defined tumor-like masses in the abdomen. It is confirmed if the evidences of bilateral chronic pleurisy and indurative mediastinitis are present.

THE DIFFERENTIAL DIAGNOSIS.—This relates to the recognition of the condition, notwithstanding the resemblance of some of its features to tumors of the stomach, liver, or other abdominal organs. To this the vagueness and irregularity of the symptoms and signs, their variations as time goes on, the primary affection, and the evidence of chronic disease in the other serous sacs all contribute. The anomalous nature of the pseudotumors and their independence of the organs may, when the effusion is not too abundant, be recognized upon careful palpation.

v. Tuberculous Peritonitis.

Tuberculosis of the Peritoneum.

The diagnosis of this condition has been fully considered under the appropriate subcaption of Tuberculosis, in the section on The Infectious Diseases. It has, in recent times, acquired peculiar importance in consequence of the remarkable success attending laparotomy in certain forms of the disease.

vi. New Growths in the Peritoneum.

Neoplasms of the peritoneum are rare. They comprise benign and malignant tumors.

1. **Benign Tumors.** — Cysts of various kinds, lipomata, fibromata, myxomata, angiomata, and other rarer forms are occasionally encountered. They may occupy any region, but are more often found in the omentum and mesentery than elsewhere. They are single or multiple. (a) CYSTS.— Cystic tumors are found in the omentum, more frequently in the mesentery. Cysts of the mesentery may be classified according to their contents into serous, chylous, hemorrhagic, dermoid, and hydatid cysts. Serous cysts are very rare. They may be single or multiple. Chylous cysts contain a milk-like opaque fluid having the characteristics of chyle, and are probably due to the retention of chyle in the lacteals, or receptaculum chyli. They have been regarded as embryonic. They are usually found in the mesentery. Hemorrhagic cysts are commonly the result of trauma, and contain a brownish-red fluid. They may be chylous or of peripancreatic origin. Dermoid cysts containing hair, bone, teeth, and mucilaginous material have been found in the omentum and mesentery. They may be multiple. Hydatid cysts usually occupy the omentum or mesentery. When primary, the cyst is commonly single. Secondary hydatid disease of the peritoneum is much more common. The cysts are usually multiple, and may be present in enormous numbers. Mesenteric and omental cysts

vary greatly in size. They may reach a capacity of several litres. (b) LIPOMATA are met with in the subperitoneal tissues of the anterior abdominal wall. They are usually small, but may attain such size as to simulate ascites. They may also develop in the omentum or in the mesentery, and grow to such a size that they completely fill the abdominal cavity. They may be of retroperitoneal origin. They occur more commonly in women, and after middle life. They are of slow growth and, yielding an obscure sense of fluctuation, suggest ascites. (c) FIBROUS TUMORS of the peritoneum are rare. They may arise from the omentum, mesentery, or the pelvic organs, and reach the size of the closed fist. Other benign tumors are exceedingly rare.

Symptoms.—Recurrent vomiting, constipation, and pain may precede the discovery of the tumor, which may occupy various positions and may be single or multiple. Mesenteric tumors are, as a rule, freely movable and may thus be distinguished from pancreatic cysts, retroperitoneal tumors, and tumors of the uterus and its appendages. An ovarian cyst with a long pedicle may, however, be very movable. Malignant tumors early contract adhesions and are usually fixed. The differential diagnosis between mesenteric and omental tumors is often attended with insurmountable difficulties. It can as a rule only be made upon abdominal section.

2. **Malignant New Growths.**—These are primary and secondary. They are of more common occurrence than the benign forms. (a) Most PRIMARY MALIGNANT GROWTHS of the peritoneum are endotheliomata. Sarcomata may occur in rare instances as primary growths starting in the mesentery and omentum. They may reach an enormous size. (b) Much more common are SECONDARY CARCINOMATA. The peritoneum is involved by metastasis from distant organs, or by direct extension from organs which it invests. The primary growth may involve the mamma, pancreas, stomach, intestines, especially the colon, and the rectum, or the uterus. In many of the cases of diffuse carcinomatous growths in the peritoneum there are the signs of an associated inflammation—*carcinomatous peritonitis.* In this form of peritonitis the exudate is usually encysted. It may consist of a yellowish serum, or a blood-stained fluid; it may be chylous, or chyliform. It is very rarely purulent. Peritoneal carcinoma is more common in middle and advanced life than earlier. It occurs with somewhat greater frequency in women than in men.

Symptoms.—Pain may be absent altogether. When present it is less severe than in other forms of peritonitis. Vomiting, constipation with attacks of diarrhœa, hiccough, and tympanites are common symptoms. Fluid exudate may be absent, scanty, abundant, freely movable, or encysted. After the withdrawal of the fluid, irregular and ill-defined tumor masses may be recognized upon palpation, especially the rolled omentum lying transversely or obliquely across the upper part of the abdomen, as a firm sausage-like growth, as in tuberculous and proliferative peritonitis. The fluid may be hemorrhagic, and contain large multinuclear cells, or groups of cells, and the number of cells showing mitosis is greater than in simple or tuberculous effusions (Dock). The temperature is usually normal or subnormal. Fever is, however, sometimes present. The cachexia may be marked, and emaciation is progressive.

Diagnosis.—Direct.—With the evidences of the primary disease, or the history of the removal of a carcinomatous breast or uterus, the diagnosis may be made without difficulty. The age of the patient, the presence of nodular masses about the navel, and enlarged inguinal glands are important. If no primary focus can be found the diagnosis may be obscure.

Differential.—The clinical resemblance to tuberculous peritonitis, as regards the symptoms, the tumor masses, and the physical signs, may be very close. As a rule, the multiple nodules of cancer are larger than those of tuberculosis. Cancer is an affection of the later periods of life, tuberculosis of the peritoneum of its earlier periods. But to this rule there are many exceptions. Inflammation and sinus formation, with discharge of pus from the navel, sometimes occurs in tuberculosis. In the absence of tuberculous disease elsewhere the diagnosis becomes difficult, since the clinical phenomena of tuberculous peritonitis not only closely resemble those of carcinomatous peritonitis, but both have features in common with the chronic proliferative form and diffuse hydatids of the peritoneum. In the last, the hydatid fremitus, and hooklets in the aspirated fluid, are of positive diagnostic value.

vii. Retroperitoneal Sarcoma.

Retroperitoneal sarcoma (Lobstein's cancer) is a rare affection. Steel finds that it occurs most frequently in the first, fourth, and sixth decades of life. Males are somewhat more commonly affected than females. The tumor may spring from the lumbar region, on the right side somewhat more frequently than the left, from the posterior wall of the abdomen near the attachment of the mesentery, or, less frequently, from the pelvis. The growths may arise from the retroperitoneal lymph-glands, the connective tissue around the vessels, or from the remains of the Wolffian body. They are often lobulated and are very prone to degeneration, with hemorrhage and the formation of pseudocysts.

Symptoms.—Vague digestive derangements and dragging abdominal pain are followed by pressure symptoms, such as neuralgic pains in the lumbar region, abdomen, legs, and genitalia, and then œdema of the lower extremities. There may be partial occlusion of the intestine. In a case of sarcoma of the retroperitoneal lymphatic glands, recently under my observation, none of these symptoms was present. There are the signs of a deep-seated tumor, situated centrally or to the right or left of the median line, sometimes moving slightly with respiration, more commonly fixed, usually solid but sometimes cystic—pseudocysts. When the tumor is situated laterally, it is obliquely crossed by the colon, which it pushes forward as it increases in size. The health is rapidly impaired, and cachexia develops.

Diagnosis.—The diagnosis rests upon the presence of the above phenomena in association with a rapidly growing central or lateral tumor about the level of the umbilicus. The differential diagnosis between retroperitoneal sarcoma and tumors arising from the kidneys and suprarenal capsules cannot always be made.

X.

THE DIAGNOSIS OF DISEASES OF THE RESPIRATORY SYSTEM.

I. DISEASES OF THE NOSE.

i. Acute Nasal Catarrh.

Coryza; Acute Rhinitis; Cold, or Cold in the Head.

Definition.—Acute catarrhal inflammation of the mucous membrane of the nasal cavities.

Etiology.—Acute nasal catarrh is very common. It is in most instances an independent affection, but it occurs also in the acute infectious diseases. It often follows exposure to cold or damp, especially when such exposure is partial, as in wetting the feet or sitting upon damp ground. It frequently prevails extensively in cold, damp, and changeable weather. Such local epidemics are to be distinguished from true influenza or grippe, to which they bear a superficial resemblance. House epidemics of coryza occasionally arise under circumstances that point to the contagiousness of the affection. Children are especially prone to it. It occurs in infants in consequence of gastric or intestinal irritation, indigestion, or the presence of intestinal worms, and is occasionally the result of injuries inflicted by foreign bodies—buttons, grains of corn, pebbles, peas, cherry-pits, and similar objects—introduced into the anterior nasal chambers. It results from the action of mechanical or chemical irritants upon the nasal mucous membrane. Among these are dust, smoke, ipecacuanha, and the fumes of ammonia, bromine, and iodine. Annoying coryza often follows the internal administration of iodine.

Coryza as a manifestation of acute constitutional infection is an early and prominent symptom of measles, influenza, and pertussis. It is sometimes associated with the ophthalmia of the new-born as the result of gonorrhœal infection incurred during parturition, and occurs as an early manifestation of congenital syphilis.

Symptoms.—The attack begins suddenly, with chilliness or shivering, a decided feeling of malaise, headache, and repeated sneezing. There is feverishness, with slight quickening of the pulse, a dry skin, and muscular pains. The nose at first feels dry and stuffy and mouth-breathing is necessary. The sense of smell is lost, that of taste greatly impaired; the voice acquires a peculiar nasal twang, and nursing infants, being unable to breathe through the nose, are suckled with difficulty. The catarrhal inflammation tends to involve the contiguous mucous tracts. In the course of a few hours from the beginning of the attack there may be a flow of thin, clear, irritating mucus, which excoriates the edges of the nostrils and the upper lip and renders the use of the handkerchief painful. Herpes labialis is common. About the second or third day the secretion becomes mucopurulent, opaque, thick, tenacious, and abundant, and tends to accumu-

late in the nasal cavities. The swelling of the mucous membrane subsides, nose-breathing is re-established, and recovery takes place within a week or ten days. Repeated attacks of the acute affection tend to produce the chronic form of the disease. Most of the cases are subacute, with symptoms of moderate intensity, little or no constitutional disturbance, and run their course in two or three days.

Diagnosis.—There is no difficulty in the diagnosis of simple acute nasal catarrh. Healthy new-born infants are not likely to suffer from snuffles. This affection, when associated with ophthalmia, is due to the same specific infection. When due to syphilis it is associated with characteristic lesions. Acute nasal catarrh in children, due to the lodgement of foreign bodies, is prolonged, and the discharge, after a time, is frequently admixed with blood. Furthermore, it is almost always one-sided. In such cases a careful examination of the nasal chambers must be made. The progress of a case of measles or influenza will speedily dissipate any uncertainty as to the nature of the acute catarrh with which each of these diseases begins. The coryza of iodism ceases upon the withdrawal of the drug.

ii. Chronic Nasal Catarrh.

Definition.—Chronic catarrhal inflammation of the mucous membrane of the nasal cavities. The cases may be arranged in three groups—rhinitis simplex, rhinitis hypertrophica, and rhinitis atrophica.

Etiology.—Repeated attacks of acute nasal catarrh may end in the chronic form of the disease. Habitual exposure to cold and draughts, a changeable and humid atmosphere, and the constant inhalation of dust are among the causes of chronic rhinitis. Insufficient food, inadequate clothing, improper ventilation, want of sunlight and fresh air, and other unhygienic conditions are predisposing influences. Chronic nasal catarrh is frequently a manifestation of local syphilitic or tuberculous processes. The nasal catarrh of early life tends to assume the atrophic form—a fact which emphasizes the importance of the prompt and efficient treatment of every case of rhinitis. Congenital asymmetry of the nasal fossæ, with marked deflection of the septum, hypertrophy of the adenoid tissue in the vault of the pharynx, traumatism, foreign bodies, and nasal polypi are local causes of chronic nasal catarrh.

Simple Chronic Nasal Catarrh (*Rhinitis Simplex.*)—This term is used to designate the transitional condition between prolonged or neglected acute catarrh and that in which hypertrophic or atrophic lesions are present. The mucous membrane is irritable and there is a constant sensation of discomfort in the nose. Catarrhal symptoms follow trifling exposure. The erectile tissue is relaxed and is readily distended with blood, so that one or both nostrils are frequently occluded. The secretion is increased; it is variable in consistency, being sometimes thin and watery, sometimes thick and tenacious. Upon inspection the mucous lining of the nasal chambers is seen to be red, watery, and irregularly swollen.

Chronic Hypertrophic Nasal Catarrh (*Rhinitis Hypertrophica*).—Obstructed nasal respiration, constant, often abundant, discharge of mucus or mucopus, frequent sneezing, nasal cough, hawking, and expectoration

of tenacious mucus, dryness of the throat, habitual mouth-breathing, especially at night, and disturbed sleep are symptoms. The voice has a peculiar nasal quality, and the hearing is very frequently impaired. In infants the inability to take nourishment without frequent interruption for respiration leads to malnutrition, and the nasal obstruction may cause attacks of suffocative spasm. In older children habitual mouth-breathing begets a peculiar, dull, facial expression, mental hebetude, and retardation of the development of the thorax, with characteristic deformities. The mucous membrane of the nasal chambers is congested throughout, and its epithelial and subepithelial tissues are hypertrophied. The characteristic lesion consists in permanent enlargement of the turbinate bodies. There is marked increase in the connective tissue with cell infiltration, dilatation of the sinuses of the erectile tissue, and loss of contractility in their walls. In a large proportion of the cases hypertrophy of the adenoid tissue in the nasopharynx and catarrhal or follicular pharyngitis occur—naso-pharyngeal catarrh.

Chronic Atrophic or Dry Nasal Catarrh (*Rhinitis Atrophica, Rhinitis Fœtidus Atrophicus, Ozæna*).—A chronic affection of the nose, constituting the terminal stage of neglected cases of rhinitis simplex and rhinitis hypertrophica. It is characterized by atrophy of the mucous membrane, with shrinkage of the turbinated bodies and diminution of the nasal secretion, which becomes mucopurulent or purulent and undergoes inspissation, with the formation of adherent and frequently offensive crusts. Upon inspection grayish crusts are seen, the removal of which exposes a smooth, pale, or a slightly excoriated, mucous surface. Actual ulceration is rare. The turbinate bodies are greatly reduced in size, their sinuses obliterated, their connective tissue contracted. The entire lining membrane of the nostrils is atrophied. The mucous membrane of the pharynx is often dry and glazed. The sense of smell is lost. Ozæna is present in a large proportion of the cases, but not in all. Odors having the same intensity and foulness are occasionally encountered in other affections of the nose attended with ulceration, as syphilis, the traumatism produced by foreign bodies, and caries and necrosis due to other causes. Atrophic rhinitis is more common in females than in males. In confirmed cases the outlook as regards cure is hopeless; as regards relief from the formation and retention of crusts and from the odor, much may be accomplished so long as a judicious treatment is persistently followed out.

iii. Autumnal Catarrh.

Hay or Rose Cold; Hay Asthma; Hay Fever; Summer Catarrh; Catarrhus Æstivus; Periodic Coryza.

Definition.—An affection of the upper air-passages, characterized by irritability of the mucous membrane, with catarrhal and asthmatic manifestations, by the abruptness of the onset of the attack, which recurs annually at or near a fixed date in the spring, summer, or early autumn, and by its immediate cessation upon the patient's reaching certain localities or upon the occurrence of frost.

Etiology. — The exciting causes are certain irritants in the atmosphere which act upon a supersensitive nasal mucous membrane in individuals of neurotic temperament. The most important of these is the pollen of various plants. In the spring and early summer the attack is caused by the pollen of certain grasses among which timothy is most toxic; in the late summer and autumn the golden rod and rag-weed are very important. The pollen of various other plants is also active in causing the disease. The sufferers show varying susceptibility to different pollens, though those who suffer from the pollen of one group of plants as the *graminaceæ* are usually sensitive to the other members of the group but are unaffected by the pollen of another group as the *Ambrosiaceæ*. The attack may be produced in sensitive persons at any season of the year by the instillation of a suspension of the specific pollen or a solution of its protein into the conjunctival sac. The endermic injection of such solutions or their application to abrased surfaces causes local or generalized outbreaks of urticaria. The condition is one of anaphylaxis. Inorganic dust of various kinds, the odors of certain flowers and other substances, emanations from animals, as the horse, and from feathers are capable of intensifying the symptoms during the attack and also of inducing similar symptoms at other seasons of the year. The intense glare of the summer sun, excessive heat, overexertion, and indigestion commonly aggravate the attack. Hay fever and bronchial asthma are not only closely associated clinically, but they also resemble each other in respect of the causes by which the attack may be excited. A similar relation between disease of the nasal mucous membrane and hay fever has also been demonstrated. There is in many cases deflection of the septum. There are often present areas of hyperæsthesia in the nasal mucosa—hyperæsthesia often so exquisite that the touch of a probe will instantly excite the characteristic train of symptoms. Males suffer more frequently than females. The disease may develop at any period in life. More than 33 per cent. of the cases begin before the age of twenty years. Dwellers in cities are especially liable to the disease, but those who live in the country do not enjoy exemption. There are certain localities in which the disease does not prevail. These regions are usually circumscribed and possess in common the attribute of an uncultivated soil. They are mostly mountainous, as certain districts in the White Mountains, the Adirondacks, and the Catskills. But elevation is not the essential factor. Relief may be experienced in any wilderness, at certain sea-shore places, on islands, or at sea.

Symptoms. — The attack makes its annual return at or about the same date. There is sometimes a period of prodromes which consist of lassitude and nervous irritability. The onset is abrupt. Itching of the palate and throat is a most annoying symptom, both common and characteristic. I have seen cases in which, year after year, this persistent itching constituted the chief local symptom. Frequent uncontrollable sneezing; nasal obstruction; free rhinorrhœa, usually thin and watery, sometimes mucopurulent; great irritation of the eyes with itching of the lids and lachrymation; loss of the sense of smell; impairment of that of taste, and not rarely disturbances of hearing, constitute the usual symptoms. These occur in paroxysms and are aggravated by changes of temperature, by sunlight, and the open air. Constitutional disturbances consist of sub-

jective sensations of heat and cold, great lassitude, complete loss of appetite, and sleeplessness. After a time the catarrh extends to the bronchi and the patient is annoyed by cough; asthmatic symptoms are common and add greatly to the distress of the sufferer. The symptoms vary in localization and in intensity, and in the same person in succeeding years. The whole duration of the attack, if not cut short by change of climate, is about six weeks. The autumnal cases usually cease abruptly upon the appearance of frost.

Diagnosis.—Sterilized pollen solutions are sold in the shops, so prepared that they may be used to differentiate hay fever from other forms of coryza and to determine the particular pollen active in any case. They are employed also for the purposes of prophylaxis and the treatment of the attack.

Prognosis.—The prognosis is favorable as regards recovery from any given attack and as regards length of life. In the absence of immunization the prognosis as regards the recurrence of the attack is much less hopeful. In some cases spontaneous desensitation eventually occurs.

iv. Epistaxis.

Nose-bleed.

Definition.— Bleeding from the nasal passages.

Etiology.— Bleeding from the nose may be due to local or constitutional causes or to a combination of both. In children its occurrence is favored by the great vascularity of the nasal mucous membrane, the frequent presence of "hemorrhagic spots," and erosions of the septum caused by picking the nose. Other local causes are chronic rhinitis, intranasal ulceration, new growths, the presence of foreign bodies, and various kinds of traumatism, especially contusions of the face.

In fractures involving the bones of the face and cranium blood may escape from the accessory sinuses or from the middle ear by way of the nose, or in hemorrhage from the lungs, œsophagus, or stomach, some part of the blood may be discharged from the nose. These blood-losses, *not from,* but merely *by way of,* the nose, do not in a strict sense constitute epistaxis—a term restricted by systematic writers to hemorrhage having its source within the nasal passages.

Among the constitutional causes are exposure to extreme cold or undue heat, or to a rarefied atmosphere, as in the ascent of high mountains and in balloon ascensions. It frequently occurs in both sexes at the age of puberty. It may result from the suppression of the menstrual flow or follow the sudden arrest of a habitual hemorrhoidal discharge. It is of frequent occurrence in anæmia in its various forms, and in persons of plethoric habit. The tendency to nose-bleed is hereditary. In hæmophilia nose-bleed constitutes a common manifestation of the hemorrhagic diathesis. It is also common in scurvy and purpura, and occurs in erysipelas, the malarial and the malignant fevers, and in nasal diphtheria. Slight nose-bleed occurs in the first week of typhoid fever with such frequency as to acquire diagnostic importance. Nose-bleed not infrequently results from the congestion and shock of the violent convulsive cough of pertussis,

It is by no means a rare symptom in advanced disease of the kidneys and in various affections of the liver. In the venous engorgement of cardiac and pulmonary diseases, even with marked cyanosis, nose-bleed is uncommon. Finally, it may result from violent mental emotion.

When epistaxis is due to general causes, the blood escapes by capillary oozing from one, rarely two or three, limited areas of the respiratory portion of the cartilaginous septum, and in most instances it proceeds from one side only. In a very small proportion of the cases it comes from the turbinate bodies or from the floor of the nostril. The mucous membrane is deeply congested, of a violaceous-red color, and shows minute spots of ecchymosis.

Symptoms.—Prodromes sometimes occur. They consist of giddiness, fulness in the head, and a sensation of dryness, tickling, or obstruction in the nostrils, which impels the patient to more or less forcibly blow the nose. More frequently these symptoms are absent, the bleeding occurring suddenly and without warning. The blood may flow in drops or for a time in a continuous stream. Ordinary, slight nose-bleed generally ceases in a short time and is without immediate clinical importance whatever may be its remote significance. The graver bleedings may be protracted for hours or days, and while a fatal case is of rare occurrence, serious consequences are likely to follow profuse hemorrhage. The arrest takes place by clotting at the point of oozing. It is important to examine the pharynx, as the clot in the nostril may lead to the escape of blood by way of the posterior nares and its being swallowed. The vomiting of blood thus swallowed may be mistaken for hæmatemesis; its expulsion by cough, for hæmoptysis, but not if due care be observed in the investigation of the case.

II. DISEASES OF THE LARYNX.

i. Acute Catarrhal Laryngitis.

Definition.—Catarrhal inflammation of the mucous membrane of the larynx.

Etiology.—"Taking cold," exposure to a cold, damp atmosphere, overuse of the voice in speaking, shouting, or singing, especially under unfavorable atmospheric conditions, as in crowded and badly ventilated halls or in the open air, are common causes of acute laryngitis. It may follow the inhalation of air charged with smoke or irritating gases or vapors. Less frequently it is due to the lodgement of foreign bodies, the action of very hot liquids or corrosive poisons, or external violence. It occurs as a local manifestation of measles, influenza, and variola, and as a complication in other acute infectious diseases, as scarlet fever, enteric fever, and erysipelas. Catarrhal laryngitis is frequently associated with catarrh of the nasopharynx and bronchi. The predisposition to largynitis varies greatly in different families and individuals.

Symptoms.—There is a sensation of dryness and tickling in the throat; the inspiration of cold air and talking cause pain. Cough is a prominent symptom. It is tickling and hoarse, or "laryngeal" in character; at first dry, later attended with scanty mucopurulent expectoration, which in

severe cases may be slightly streaked with blood. The voice, at first husky, grows rapidly hoarse, and at length may be completely lost. Dyspnœa is not common in adults, but it is a very frequent symptom in early life, usually occurring in paroxysms and at night. In severe cases cough is very harassing, deglutition is painful, and there may be urgent dyspnœa. Laryngoscopic examination shows that the mucosa is reddened and swollen, especially between the arytenoid cartilages and in the aryepiglottic folds. When the inflammation is intense the vocal cords present superficial erosions, and minute hemorrhages are seen at various points of the laryngeal mucous membrane. A scanty exudation of altered mucus is irregularly scattered upon the surface. In phonation there may often be observed imperfect approximation of the vocal cords, due to implication of the intrinsic muscles of the larynx in the inflammatory process.

The constitutional symptoms vary; they are not usually severe. Moderate fever, with headache and loss of appetite, may occur. The attack lasts from a few days to a week or more and terminates in recovery. Neglected cases may assume the chronic form.

ii. Acute Laryngitis of Children.

Spasmodic Croup; False Croup.

The special feature consists in paroxysmal exacerbations, suffocative in character and occurring at night. These are due to the relative smallness of the larynx in infancy, the narrowness of the rima, the looseness and vascularity of the mucous membrane, and the greater reflex excitability of the nervous system. The disease is a common one, occurring with frequency during the first dentition, and particularly during the second and third years.

Etiology.—Exposure to cold and damp, chilling of the surface, violent screaming, the inhalation of steam, smoke, and dust, and indigestion are causes of acute laryngitis in infants. It occurs more frequently in the cold, damp months of winter and spring than in the summer and autumn. It is somewhat more common in male than in female children, and certain families and individuals manifest an especial liability.

The attack may come on abruptly or be preceded by fretfulness, loss of appetite, and trifling elevation of temperature, huskiness or complete aphonia, and a harsh, croupy cough. Inspiration is prolonged and stridulous; there is recession of the suprasternal and supraclavicular spaces; the pulse is frequent and small, and the lips and finger-tips are cyanotic. There is great restlessness, and the expression indicates anxiety and distress. The attack presently passes off, either spontaneously or after the administration of simple remedies. The child presently falls asleep again and rests until morning; or the attack may be repeated once or several times in the course of the night. On the following day he scarcely seems ill and plays about as usual, but toward evening the croupy cough reappears and during the night the attacks of croup occur as before, to be again repeated, as a rule, upon the third and rarely upon the fourth night, but with diminishing severity. After that there remains simply a trifling bronchial catarrh, which in the course of a few days disappears.

Diagnosis.—Direct.—Acute laryngitis of the adult rarely presents difficulty in diagnosis. The severer cases suggest œdema of the larynx—acute laryngeal œdema—while those attended by complete loss of voice may be mistaken for hysterical aphonia or paralysis of the vocal cords due to other causes. These questions are at once settled by the laryngoscope.

Differential.—In children the diagnosis of acute laryngitis is, in certain cases, attended with serious difficulty. The condition is to be distinguished from laryngismus stridulus by the presence of fever, the catarrhal symptoms, the mode of onset, the character of the paroxysms, their nocturnal occurrence, the hoarseness and loss of voice, the absence of the prolonged crowing inspiration which terminates the attack of laryngismus, and the course and duration of the disease.

The diagnosis between spasmodic croup and laryngeal diphtheria—*membranous croup*—may be for a time impossible. The principal points in favor of spasmodic croup are the milder character of the constitutional symptoms which precede the signs of laryngeal obstruction, the paroxysmal nature of the obstruction, and the complete relief between the attacks, the progressive amelioration of the symptoms after the second night, the absence of exudation upon the tonsils and adjacent parts, and the absence of enlargement of the cervical nodes.

Prognosis.—The outlook is favorable. The most alarming symptoms, as a rule, promptly subside after the emesis caused by ipecac, or after a warm bath and the proper administration of simple sedative remedies.

iii. Subacute Laryngitis.

By far the larger number of cases of catarrhal laryngitis are of the mildest type. The patients are not ill; the only symptoms are a slight tickling cough, with hoarseness or aphonia.

The condition acquires importance from its great relative frequency; from the fact that, being accompanied by trifling subjective symptoms, it is likely to be neglected; and, finally, because in many cases prolonged, habitual exposure to the original cause, or use of the voice when the larynx is slightly congested or inflamed, convert a passing local indisposition into a serious disease. In fact the larger proportion of cases of chronic laryngitis arise in this way.

iv. Chronic Laryngitis.

Etiology.—This form may be the sequel of an acute attack; more commonly it is the result of the persistent action of causes which give rise to subacute catarrh. Improper use of the voice and its habitual overuse in singing, public speaking, or shouting in the open air are very common causes of chronic laryngitis. It is sometimes associated with chronic pharyngitis and especially with that form which is caused by habitual overindulgence in alcohol and tobacco, with certain cases of marked obstruction to nasal respiration, and cases of elongation of the uvula. Chronic laryngitis is more common in males than in females and is especially a disease of middle life.

Symptoms.—There is a tickling sensation in the throat accompanied by a desire to obtain relief by coughing. As a rule pain is not present except after prolonged use of the voice or coughing. Many patients complain of a disagreeable feeling of dryness. The voice is rough and hoarse and at times almost lost. The cough is ringing, loud, deep; expectoration is as a rule scanty and tenacious, but occasionally abundant and sometimes fetid. Upon laryngoscopic examination the mucous membrane is found irregularly thickened and discolored, but the redness is less intense than in the acute form. The vocal cords are of a grayish-red color, and in debilitated and cachectic persons there may be seen minute superficial erosions. The epiglottis is in many cases irregularly thickened. The general health is often impaired.

Diagnosis.—The local sensations, chronic alteration of the voice, and peculiar cough suggest the true nature of the affection, but a positive diagnosis can be made only after careful laryngoscopic examination. In every case of chronic laryngitis the history of the patient in all particulars must be carefully investigated in order to determine whether or not the local affection be primary, or secondary to some other disease, as alcoholism, tuberculosis or syphilis.

v. Œdematous Laryngitis.

Acute Laryngeal Œdema; Œdema of the Glottis.

Etiology.—Œdema of the mucous and submucous tissues of the larynx occasionally occurs as a serious and frequently fatal complication in the course of acute catarrhal laryngitis, whether due to cold or to internal or external traumatism; in chronic disease of the larynx, as tuberculosis and syphilis; in connection with perichondritis of the larynx; as a complication of severe inflammatory affections of neighboring structures, as the tonsils, parotid glands, or the cellular tissue of the neck; in the course of acute infectious diseases, as scarlatina, typhoid fever, variola, and erysipelas; and, finally, as an extension of the general œdema in acute or chronic nephritis.

Symptoms.—Rapidly progressive dyspnœa is the chief symptom. It is at first inspiratory; later also expiratory. Respiration is accompanied by loud stridor. The voice becomes husky and soon fails. Signs of impending suffocation supervene, and unless relief is afforded death takes place in the course of a few hours. If a laryngoscopic examination prove successful, the epiglottic and aryepiglottic folds are seen to be greatly swollen, the latter almost meeting laterally; the false cords are also œdematous. These changes can be felt with the finger, and upon depressing the tongue the swollen rim of the epiglottis may sometimes be brought into view.

The diagnosis is unattended with difficulty and depends upon physical exploration.

vi. Pseudomembranous Laryngitis.

True Croup; Membranous Croup; Fibrinous Laryngitis.

Definition.—Inflammation of the mucous membrane of the larynx, resulting in the formation of a pseudomembrane or pellicle composed of a network of fibrin, embracing in its meshes leucocytes and necrotic epithelium.

Etiology.—Any agent capable of destroying the protecting epithelium of the laryngeal mucous membrane, thus permitting the escape of serum and white blood-corpuscles, may give rise to the formation of a pseudomembrane. Hence, this form of laryngitis may result, (a) from traumatism, as the inhalation of steam, hot smoke, or irritating and corrosive chemicals in the form of vapor or solution; (b) from the action of certain pathogenic micro-organisms.

Pseudomembranous laryngitis occurs at all seasons of the year. It especially affects young children between the ages of two and six. Cases in children under two and over seven years of age are much less common. Exceptionally the disease occurs at a later period of life. Boys are somewhat more liable than girls. This affection frequently occurs as a complication in scarlet fever and measles. In by far the greater number of cases it is a manifestation of diphtheria.

Symptoms.—The symptoms usually develop in the course of an attack of faucial diphtheria or of one of the exanthemata. Less frequently they arise as the manifestations of a primary laryngeal diphtheria. They point to progressive impairment of the functions of the larynx, with increasing obstruction to respiration and its consequences, and consist of hoarseness, aphonia, explosive and croupy cough, stridulous respiration, dyspnœa, recessions, restlessness, cyanosis, and stupor.

Diagnosis.—Acute progressive laryngeal stenosis in a young child is nearly always due to pseudomembranous laryngitis. If traces of the exudate can be discovered upon inspection of the throat, or if, upon physical examination, there can be detected coarse or whistling tracheal râles, or finally, if shreds of membrane are expectorated after paroxysms of explosive cough, the diagnosis becomes sure. It is equally so, in the absence of such confirmatory evidence, if the case occur in a locality already the scene of an epidemic of diphtheria. The fact that, even in pseudomembranous laryngitis, the signs of obstruction are at first paroxysmal and followed by intervals of partial relief must always be borne in mind. For this reason the early differential diagnosis between this disease and spasmodic laryngitis is not, in all instances, possible. In the latter, however, the intervals of relief are more complete and prolonged, the paroxysm not usually recurring until the succeeding night; the tendency is to progressive amelioration of the symptoms rather than progressive aggravation, and the signs of grave constitutional disturbance do not show themselves.

Prognosis.—Pseudomembranous laryngitis is, in the absence of treatment, an extremely fatal disease. The diphtheritic form, under the administration of antitoxin, frequently terminates in recovery. It is therefore imperatively necessary to at once employ this remedy.

vii. Tuberculous Laryngitis.

Laryngeal Phthisis; Throat Consumption.

Definition.—Inflammation of the tissues of the larynx caused by local tuberculosis.

Etiology.—Tuberculosis of the larynx may occur as a primary disease. Much more frequently, however, it is secondary to pulmonary tuberculosis. When the earliest symptoms are laryngeal, the disease remains for a time localized, but eventually the lungs become involved. Secondary tuberculous laryngitis occurs in more than 25 per cent. of the pulmonary cases. The laryngeal symptoms are pronounced and the lesions extensive and advanced in a much smaller percentage. A majority of the cases occur in males—a fact attributed to their greater liability to chronic catarrhal laryngitis, which acts as a predisposing cause. Not every case of chronic laryngitis in a consumptive individual is tuberculous. The mechanical irritation of frequent and severe cough and the contact of the sputum may cause chronic catarrhal laryngitis, which is aggravated by the condition of the patient, and which undoubtedly, after a time, predisposes to infection. In the tuberculous cases the mucous membrane is of a grayish, pale color, irregularly mottled and congested; it is at first swollen and studded with miliary tubercles, which by their coalescence form scattered tuberculous nodules. These nodules undergo caseation, as a result of which there form more or less extensive superficial ulcers, which show a tendency to spread. The floor of these ulcers is covered by a grayish exudation, and they are surrounded by a border of infiltrated and swollen tissue. They occur most frequently upon the arytenoids, in the interarytenoid space, upon the true cords, and on the epiglottis. The destruction of tissue extends deeply, implicating the submucosa, and in severe cases the perichondrium and cartilages, which undergo more or less extensive necrosis—tuberculous perichondritis and chondritis. The ulcers occasionally extend to the back of the tongue, to the pharynx, to the upper part of the œsophagus, and in severe cases to the pillars of the fauces and the tonsils. Complete erosion of the true cords not infrequently occurs, and the epiglottis is often destroyed throughout the greater part of its extent.

Symptoms.—The earlier symptoms are those of chronic laryngitis due to other causes. There is slight huskiness, which is at first intermittent and disappears after resting the voice. It soon becomes continuous, and gives place to a peculiar hoarseness, which in the advanced stages of the disease usually passes into complete aphonia. Cough is tickling, paroxysmal, and unproductive; it has the peculiar quality known as laryngeal, and may be distinguished in the same patient from the mere nervous cough of bronchial irritation. It is not at first distressing, but in cases of advanced ulceration it becomes husky and high-pitched, and is attended with pain. Spontaneous pain is not very common. There is often tenderness upon external pressure. Dysphagia is a prominent and most distressing symptom in advanced cases, especially when the epiglottis is involved, the arytenoids are extensively destroyed, or there is ulceration of the pharyngeal wall. In such cases the administration of nourishment is

attended with difficulty, the attempt to take food of any kind giving rise to severe pain, urgent paroxysms of cough, and frequently to suffocative attacks. The difficulty in swallowing adds greatly to the sufferings of the patient and constitutes the most distressing symptom of the terminal stage of this form of tuberculosis. In the earlier stages the laryngoscope reveals the appearances due to chronic laryngeal catarrh. There is, however, greater pallor of the mucous membrane, together with some thickening over the arytenoids. Later the picture is characteristic. The vocal cords are thickened and eroded, and their motility is impaired; the epiglottis and arytenoid are infiltrated, and at various points superficial grayish ulcers with ill-defined borders are seen; finally, deep ulceration, with extensive loss of substance, occurs.

Diagnosis.—Direct.—In the earlier stages, especially in the absence of the evidences of pulmonary tuberculosis, the diagnosis of tuberculous laryngitis cannot always be made. Pallor of the laryngeal mucous membrane, thickening of the arytenoids, general failure of health on the part of the patient, and absence of response to local and constitutional treatment lead to the suspicion of tuberculous disease. This suspicion is confirmed by the appearance of the characteristic ulceration, the evidences of pulmonary tuberculosis, or the detection of tubercle bacilli in the sputum or the exudate scraped from the floor of the laryngeal ulcer. In selected cases the tuberculin tests may be made.

Differential.—The diagnosis between tuberculosis and syphilis of the larynx is, in certain cases, attended with some degree of difficulty. In this connection, the greater tendency of syphilis to invade the pharynx, the fact that tuberculous ulceration of the larynx is, in general, progressive and continuously destructive, while syphilitic ulceration frequently shows a disposition to heal at one point while advancing at others, and, finally, the history of specific inflammatory or ulcerative lesions in other parts of the body in syphilis should receive due consideration.

Prognosis.—The course of tuberculous laryngitis is, as a rule, in the highest degree unfavorable. While, in the literature of the subject, cases of marked amelioration, or even of cure, especially in the primary form, are reported, the disease is so constantly fatal that the instances in which more than a temporary arrest occurs must be regarded as exceptional.

viii. Syphilitic Laryngitis.

Definition.—Inflammation of the larynx, occurring as a manifestation of syphilis, either hereditary or acquired.

Etiology.—The larynx is very frequently involved in syphilitic inflammation.

The catarrhal laryngitis of secondary syphilis presents nothing characteristic. Symmetrical superficial ulceration of the true and false cords occurs. Mucous patches, when present elsewhere, confirm the diagnosis, but they are not common in the larynx.

Much more frequent and important are tertiary lesions. Gummata, multiple or single, develop in the submucous tissues. They may undergo resolution, or, as is much more frequently the case, they break down,

giving rise to extensive and deep ulceration, which may involve the cartilages. Sometimes the disease begins as a perichondritis attended with suppuration, and rapidly causing necrosis of the cartilages. In such cases external fistulæ may be formed. In the course of the ulceration, erosion of the walls of arterial branches may give rise to free hemorrhage, or an acute œdema may prove rapidly fatal. The gummata develop most commonly at the base of the epiglottis or in the ventricles. They may attain the size of a nut and occasion serious stenosis of the larynx. The sclerosis which attends their resorption, or the cicatrices resulting from the healing of the ulcers, are often the occasion of marked deformity of the larynx, with progressive stenosis.

The gummatous infiltration of inherited syphilis in either the early or the later form leads to ulceration, which tends to extend deeply and involve the cartilages. The healing of such ulcers is also likely to be followed by cicatricial stenosis and deformity.

Symptoms.—Secondary syphilis of the larynx gives rise to hoarseness and laryngeal irritation. The symptoms of the tertiary lesions are of the most serious character, consisting during the stage of active ulceration of aphonia, cough, pain, dyspnœa, dysphagia, and in the stage of cicatrization of a more or less grave and progressive mechanical obstruction to respiration. The symptoms show themselves in the hereditary disease commonly within the first six months of life; exceptionally, after puberty.

Diagnosis.—The history of the case and of other specific cutaneous inflammatory or ulcerative lesions, or the presence of such lesions or their scars, renders the diagnosis in a majority of the cases a simple matter.

Prognosis.—Under early and prolonged antisyphilitic treatment the outlook is favorable. With the general improvement the laryngeal symptoms subside. In old cases with stenosis, tracheotomy may be necessary.

ix. Laryngismus Stridulus.

Definition.—A neurosis, the prominent symptom of which is spasmodic closure of the glottis, associated, in severe attacks, with spasm of the diaphragm and other muscles of respiration. The relaxation of the spasm is accompanied by a prolonged, high-pitched, crowing inspiratory sound, from which the affection receives its name.

Etiology.—Laryngismus stridulus occurs almost exclusively before the end of the third year of life. It is more common in boys than in girls. A large proportion of the cases occur in rhachitic children, but those in fair health may develop the attack without warning. The paroxysm may be excited by a variety of causes, either physical or emotional. Among these are sucking, sudden movements, violent crying, the bath, indigestion, and dentition. They also occur in the absence of such causes—on waking from sleep, for example—and more frequently by night than during the day. The seizure may present, especially in older children, a curious appearance of being voluntary, and is sometimes regarded at first as a fit of passion or of holding the breath.

Symptoms.—The attack may be preceded by an occasional catch in the breath or by slight crowing sounds; as a rule it comes on without

premonitory symptoms. There is complete arrest of respiration. The chest is fixed, the head thrown back, the face, at first pale, quickly becomes cyanotic, the eyes are wide open and staring. There is often twitching of the facial muscles. In the severer cases there may be opisthotonos, carpopedal spasm, or general convulsions. The attack lasts from a few seconds to a minute or more. Death has taken place during the paroxysm from prolonged stoppage of respiration or from impaction of the epiglottis. As the cyanosis deepens the spasm yields; the air slowly enters the lungs again through the relaxing glottis, with the characteristic prolonged, high-pitched, crowing sound, and the attack ends in a spell of coughing or crying. The seizures vary greatly in severity and number. After a few repetitions they may cease altogether, or they may come on very frequently both by day and by night, and recur during a period of months.

Diagnosis.—The absence of fever, hoarseness, and cough in the intervals between the attacks, the suddenness and completeness of the arrest of breathing, the short duration of the paroxysm, the peculiar prolonged crowing inspiration with which it ends, and the associated convulsive phenomena, taken together, form a characteristic clinical picture not to be mistaken for any other malady. When death occurs in the paroxysm the crowing is absent, and the sudden asphyxia may remain unexplained.

Prognosis.—As regards the spasm the outlook is favorable, the fatal cases being few in number. Children who suffer from laryngismus stridulus are, as a rule, frail, and a large proportion of them succumb to intercurrent disease.

x. Chronic Infantile Stridor.

Definition.—The chief symptom consists of an almost continuous coarse, low-pitched, inspiratory stridor, which is present both when the child is awake and during sleep.

Etiology.—The cause is unknown.

Symptoms.—The stridor varies in intensity, being much aggravated by excitement. It sometimes ceases wholly for a few hours. As the disease gradually passes off, it occurs only at intervals and when the child is lively or excited. Expiration is usually normal; it may be accompanied by a few coarse mucous râles. Retraction of the thorax does not often occur, and when present is slight. In one case only have I encountered faint cyanosis, and in that instance there were, during the eighteen months of stridulous breathing, three transient general convulsions. The case ended in recovery. As a rule the affection does not seem to interfere with the general health of the child.

xi. Paralysis of the Laryngeal Muscles.

The larynx is supplied by the superior laryngeal and inferior or recurrent laryngeal branches of the vagus. These are joined by branches of the sympathetic. The superior laryngeal nerves supply the mucous membrane of the upper portion of the larynx, including the epiglottis, as far as the true cords. They also supply the cricothyroid, the thyro-epiglottic and the aryteno-epiglottic muscles, and the arytenoid muscles which also

derive motor filaments from the recurrents. The inferior or recurrent laryngeals curve around the arch of the aorta on the left side and the subclavian on the right, and ascend between the trachea and œsophagus to supply the laryngeal mucous membrane below the cords and all the muscles of the larynx except the cricothyroids. The superior and inferior laryngeal nerves on each side communicate with each other in two places, namely, at the back of the larynx and on the side of the larynx under the ala of the thyroid cartilage. The motor filaments of these branches of the vagus are derived from the spinal accessory.

In paralysis of the laryngeal muscles the lesion may be:

1. Central, involving the nucleus of the accessory nerve in the medulla. The laryngeal paralyses of this group arise as a result of syphilis affecting the medulla oblongata, acute and chronic bulbar paralysis, multiple sclerosis, and locomotor ataxia. The hysterical paralyses of the larynx must also be regarded as of cerebral origin.

2. The lesion may affect the fibres of the recurrent laryngeal in the course of the vagus or the accessory nerve. This group includes the cases in which the paralysis is due to pressure by new growths, and there are cases in which the trunk of the nerve is wounded or injured in surgical operations above the point at which the recurrents are given off.

3. The lesion may directly involve the laryngeal nerves. The majority of the cases of laryngeal paralysis are included in this group. The recurrents are, by reason of their remarkable course, especially liable to abnormal pressure by new growths, both within the thorax and in the neck. The left, which curves around the aorta, is exposed to greater risk of injury than the right, which passes no lower in the chest than the subclavian. Either may be included in the dense pleural thickening at the apices which occurs in certain forms of pulmonary tuberculosis. Paralysis of the right is in rare instances caused by aneurism of the subclavian artery. The left is likely to be injured by the pressure of an aneurism of the arch of the aorta, a mediastinal tumor, enlargement of the bronchial glands, and in rare cases of a massive pericardial effusion. Both, as they ascend between the trachea and the œsophagus, are occasionally involved in carcinoma of the latter, or compressed by enlargement of the thyroid gland. Paralysis of the recurrents occurs as a very rare sequel of diphtheria and as a result of chronic alcoholism.

4. The lesion may be confined to the larynx. The loss of power is purely muscular and amounts merely to a paresis. This occurs in various diseases, and is due to inflammatory infiltration of the submucous tissues with altered nutrition of the muscles.

5. Finally, cases of laryngeal paralysis occur for which no adequate cause can be discovered.

The following are the more important forms of laryngeal paralysis:

1. Complete Paralysis of the Recurrent Nerve.—This condition occurs as the result of lesions dividing or completely destroying the recurrent or its fibres in the vagus, or as a manifestation of neuritis due to diphtheria or other causes, or in consequence of advanced disease in the medulla. It may be unilateral or bilateral. When the paralysis is unilateral, the vocal cord on the affected side occupies the median or so-called cadaveric

position, and is motionless upon inspiration, expiration, and attempts at phonation. In phonation the vocal cord and the arytenoid of the sound side pass beyond the median line. The voice is harsh, it easily breaks into a falsetto, and speaking is attended with effort. The cough is likewise harsh and brassy. Dyspnœa is not a symptom. In complete bilateral paralysis—a very rare condition—the cords occupy the median position and are immobile; their edges are slightly concave, as the aperture is sufficiently wide for respiration; dyspnœa is absent except upon exertion. Aphonia is complete and coughing is impossible.

2. Bilateral Paralysis of the Abductors.—The posterior crico-arytenoids are involved. This form of laryngeal paralysis may occur as a central affection in the course of bulbar paralysis, multiple sclerosis, and locomotor ataxia. It may be produced by pressure upon both vagi or upon both recurrents. It is encountered as a rare form of hysterical palsy. Abductor paralysis may follow exposure to cold or may arise in the course of a laryngeal catarrh. The cords are approximated as in phonation. The glottis is not opened in inspiration; on the contrary, it acts like a valve, and is narrowed by the pressure of the air to a small slit. Inspiration is therefore difficult, prolonged, and stridulous, while expiration is unimpeded. Phonation is not affected. The ability to cough remains. This form of laryngeal paralysis is rare, but is attended with the danger of sudden suffocation. If the symptoms are progressive and the dyspnœa constant, tracheotomy becomes necessary and the tube must be constantly worn.

3. Unilateral Abductor Paralysis.—One cord only may be affected in pressure-paralysis involving the recurrent of one side. Aneurism of the arch of the aorta, exerting pressure upon the left nerve, is by far the most common cause of this condition. The right nerve is especially liable to be involved in pleural thickening and retraction of the apex of the lung in the course of pulmonary tuberculosis. The vocal cord on the affected side remains fixed in the middle line during inspiration. The voice is sometimes unaffected; more commonly it is slightly harsh or rough. Dyspnœa and stridor are not often present. The movements of the other cord are normal.

4. Adductor Paralysis.—In the more common forms of adductor paralysis the lateral crico-arytenoids, the arytenoid, and the thyro-arytenoids, are implicated. It occurs as the result of exposure to cold or from overuse of the voice, and is very often the cause of loss of voice in catarrhal laryngitis; it is the usual form of paralysis in hysterical aphonia. The laryngoscope reveals the normal position and movement of the cords in respiration but their total failure to approximate on attempts at phonation. There is neither stridor nor dyspnœa; ability to cough is not affected, but aphonia is complete. Adductor paralysis may be partial. It is commonly bilateral, but in exceptional cases unilateral. In bilateral paresis of the thyro-arytenoids the glottis does not close completely on phonation, the margins of the cords being separated by an oval space. If one cord only is affected its margin remains concave. In paralysis of the arytenoid, which seldom occurs alone, the vocal cords are brought together in their anterior extent, but the failure of the arytenoid cartilages to approximate leaves a narrow triangular opening at the interarytenoid space.

III. DISEASES OF THE BRONCHI.

i. Bronchitis.

Definition.—Inflammation of the whole or any part of the bronchial mucous membrane. It occurs as an acute or chronic disease. It is bilateral and usually limited to the larger or medium-sized tubes. When it extends to the smaller and terminal bronchi it is spoken of as "capillary bronchitis," but this condition is always associated with collapse and inflammation of the corresponding air-vesicles, constituting bronchopneumonia.

(a) ACUTE BRONCHITIS.

This very common affection is not often serious in the middle periods of life. In infancy and old age it tends to involve the smaller tubes and is often a fatal disease.

Etiology.—Chilling of the surface, and especially wet feet, tend to produce engorgement of the bronchial vessels and the microbic infection to which bronchial catarrhal inflammation is due. Overheated dwellings, a damp or dust-laden atmosphere, and, in rare instances, the inhalation of irritating gases—chlorine, bromine, etc.—are also etiological factors. Certain persons suffer from a peculiar susceptibility and develop the disease upon slight exposure to its causes. It very often arises as the extension downward of an ordinary coryza, the result of "catching cold," and is common in damp, cold, and changeable weather, when it often prevails in local epidemics. Acute bronchitis constitutes an important element in measles, pertussis, and asthma, and is frequently met with in the ague fit of malaria, and early in the course of enteric fever.

Pathology.—The significance of the clinical phenomena rests upon the anatomical changes, which in the main consist of redness and congestion of the mucosa, swelling and œdema of the submucosa, infiltration of the tissues with leucocytes, desquamation of the epithelium in its ciliated and embryonic forms, and the secretion of mucus and pus.

Symptoms.—The onset is frequently characterized by symptoms of constitutional infection, chilliness, crawling sensations, fever,—101°–103° F. (38.5°–39.5° C.),—bodily and mental depression, languor, and pains in the back and limbs. There are sensations of substernal pain and constriction, a rough, dry, and sometimes ringing cough, often paroxysmal and distressing, and much uneasiness and pain in the chest, especially along the insertions of the diaphragm. In the course of a day or more the cough loosens, with much relief of the respiratory symptoms and disappearance of the fever and other evidences of constitutional trouble. The expectoration becomes free, abundant, and mucopurulent, and later purulent and nummular.

Physical Signs.—Bronchitis of the larger tubes may yield no abnormal physical signs. The percussion sound is not altered in an uncomplicated acute bronchitis. Upon auscultation in the early stage dry râles, sonorous and sibilant, are heard at various points on both sides of the chest. They vary in size and quality, often disappearing after efforts of coughing.

When the cough becomes loose and the expectoration fluid and abundant the râles become moist and bubbling. Rhonchal fremitus is often present, especially in children. The respiratory murmur is vesicular, never bronchial. If the bronchial secretion is very abundant, there may be slight temporary dyspnœa and enfeeblement of the vocal fremitus, both of which disappear after cough with free expectoration. The intensity and course of the disease are variable. The attack in many cases scarcely amounts to an illness. Adults in previous good health usually recover in the course of a few days, the fever subsiding by rapid lysis, and cough and expectoration gradually diminishing.

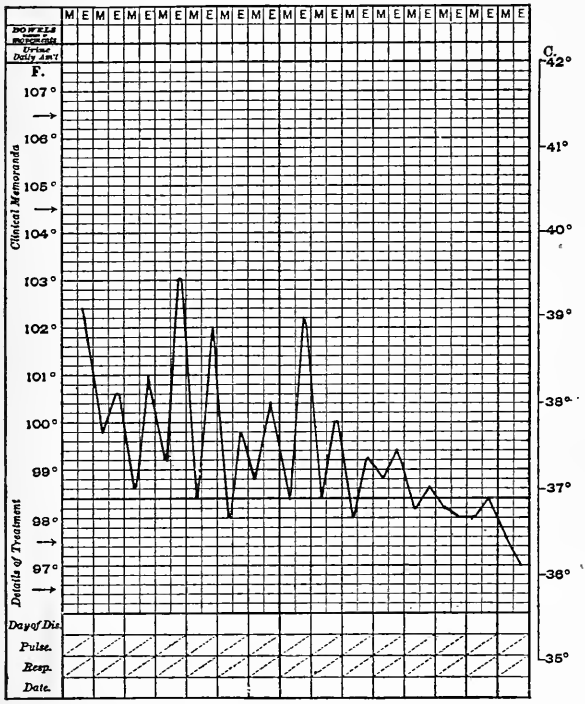


FIG. 317.—Acute bronchitis. Woman aged 50.—German Hospital.

Diagnosis. — In infants, the aged, and debilitated persons at all periods of life, there is danger that the bronchial catarrh may invade the fine tubes and cause bronchopneumonia. A daily examination should be made as a matter of routine. It is important alike from the standpoint of diagnosis, prognosis, and treatment to note whether the râles are dry or moist, since these qualities are indicative of the physical characters and amount of bronchial secretion, and whether they are coarse, medium-sized, or small, since variations in this respect correspond to variations in the diameter of the tubes involved. Subcrepitant and crepitant râles at the bases posteriorly, together with faint vesiculobronchial breathing and relative dulness, are the signs of extension to the finer tubes. With these signs there is a rise of temperature, increased respiration and pulse-frequency, restlessness, slight cyanosis, and the general appearance of an aggravation of the illness.

In isolated cases of measles the differential diagnosis cannot be made until the appearance of Koplik's sign or the exanthem; in pertussis it cannot be affirmed until the whoop comes, though cough in paroxysms which cause vomiting and are worse at night is suggestive. Bronchitis is by no means rare in the early stage of enteric fever, and there are occasional cases in which for a few days cough and expectoration are prominent symptoms.

(b) CHRONIC BRONCHITIS.

The affection very rarely arises as the termination of a single attack of acute bronchitis. It sometimes follows the repeated attacks which result from continuous exposure to the cause of catarrhal affections.

Etiology.—Chronic bronchitis is a secondary disease. It constitutes an important manifestation of certain circulatory derangements, as heart disease, thoracic aneurism, arteriosclerosis, some chronic pulmonary affections, as pneumoconiosis, asthma, and emphysema, and constitutional conditions, as gout and chronic uræmia. Important predisposing influences are climate and season. The winter cough of elderly and invalid persons is well known and is often absent when the patients are able to avoid the cold and changeable weather of the north by a temporary stay in a warm, dry, and equable climate. The influence of age is marked. Acute bronchitis is a disease of the young; chronic bronchitis a disease of the old. Chronic bronchitis is more common in men. It sometimes comes on in women at an early age without obvious cause and runs an indefinite course, with mild symptoms and slight secondary changes—bronchiectasis and emphysema.

Pathology.—The lesions are not evenly distributed. They affect different parts of the bronchial mucosa in varying degree and in irregular patches, and consist of loss of epithelium, atrophy of the glands and muscularis, thinning of the mucous membrane, and dilatation of the walls of the tubes. Bronchiectasis and emphysema gradually come to pass.

Symptoms.—There is cough of variable severity, less troublesome in dry warm weather and always worse in the cold and changeable weather of winter and early spring. There are often paroxysms in the morning, with comparative freedom throughout the day. In some cases cough is especially troublesome at night. The sputum has no constant characters. It differs in different cases and at different times in the same case. In the dry catarrh expectoration is absent. As a rule it is abundant, coming up in considerable quantities at a time. Sometimes there is a little tenacious mass of clear mucus at intervals. Very common is a clear, thin fluid. In the advanced cases shortness of breath is common upon exertion. It is due to emphysema and to some extent also to cardiac weakness. Fever is absent. There is no pain. The general health is often good, and the patients may be fully able to attend to their affairs. The disease is, however, progressive, and tends to an ultimate dyscrasia with advanced emphysema, bronchiectasis, and dilatation of the right heart.

Physical Signs.—In the early stages the physical signs do not differ from those of the stage of expectoration in acute bronchitis. Already perhaps the percussion sound has the vesiculotympanitic quality. There is prolongation of the expiratory sound, and various râles are heard, some sonorous, some sibilant, and occasionally moist râles of every size. At the bases posteriorly there are heard subcrepitant and crepitant râles, and when there is much fluid secretion there may be slight impairment of resonance.

Clinical Varieties.—(1) *Dry Bronchitis; Catarrhe Sec.*—This form is characterized by troublesome, paroxysmal cough, with slight expectoration, sometimes none at all. It is not uncommon in old people with emphysema.

(2) *Bronchorrhœa.*—The expectoration is profuse, two or more pints sometimes being brought up in the course of twenty-four hours. It may be thin and watery —*bronchorrhœa serosa*—or thick and uniformly purulent, or, and this is most common, it may consist of a thin pus with greenish clumps. *Bronchorrhœa* is to be distinguished from bronchiectasis, to which, by soakage and the dilating pressure of the accumulating secretion, it tends to give rise. The retained fluid may undergo decomposition.

(3) *Putrid Bronchitis.*—Foul-smelling expectoration may occur in a number of different conditions. These comprise anatomical lesions in which secretions are retained, as bronchiectasis, vomicæ, empyema with bronchopulmonary fistula, and pulmonary abscess. In addition to these, there is a special form of bronchitis characterized by sputum having the odor of decomposition. This form is comparatively rare. The expectoration is abundant, of a dirty yellowish-gray color, thin, and separates upon standing into three layers, an uppermost, greenish-yellow in color, consisting of thin froth, a middle transparent serous layer, and an opaque, purulent sediment, in which may sometimes be found small, whitish-gray masses—Dittrich's plugs. Putrid bronchitis may be followed by aspiration pneumonia, abscess, or gangrene. It can be differentiated from bronchiectasis only when the latter forms cavities sufficiently large to yield characteristic physical signs, the sputa being practically the same in the two conditions; from vomicæ by the presence of tubercle bacilli in the sputum, and the signs of a cavity, usually in the neighborhood of an apex; from gangrene by the presence of shreds of necrotic pulmonary tissue, and finally, from empyema communicating with a bronchus, by the more distinctly purulent sputa, its expectoration in larger quantities at a time, the greater readiness with which it is brought up when the patient lies upon one—the unaffected—side, and the physical signs, which indicate a unilateral and circumscribed lesion.

(c) FIBRINOUS BRONCHITIS.

Plastic or Croupous Bronchitis.

Bronchial casts are occasionally found in infralaryngeal diphtheria, in croupous pneumonia, in chronic valvular disease of the heart, and in the *stadium ultimum* of pulmonary tuberculosis. They have also been found in the copious albuminous expectoration which, in very rare instances, supervenes upon the removal of a pleural effusion by aspiration. After hæmoptysis branching blood-casts are frequently expectorated. All these conditions are to be distinguished from fibrinous bronchitis, a form of bronchitis characterized by the formation, in limited branches, of casts or moulds of the bronchial tubes which give rise to urgent dyspnœa and are expelled in violent paroxysms of cough. There are acute and chronic forms, without direct etiological relationship. *Acute Fibrinous Bronchitis.*—This form usually occurs as an intercurrent affection in the febrile infectious diseases. There is bronchitis with increasing dyspnœa; in some cases a rise in temperature and chills have been noted. Casts are coughed up. They are usually arborescent, sometimes merely a straight short

mould of a single tube, with its terminal subdivisions. In fatal cases the casts have been found in the tubes. *Chronic Fibrinous Bronchitis.*—This form occurs as a primary affection. It is a rare disease. It is more common in middle life and in men than in women. The exciting cause is unknown. The attacks recur at intervals with more or less regularity for long periods of time. There are signs of bronchitis, with fever. Hæmoptysis occurs. Dyspnœa is marked. Cyanosis may be present. The physical signs may be localized or, if diffuse, they are intensified over the affected area. There is enfeeblement or absence of the respiratory murmur, without impairment of resonance, together with many râles on coughing. The respiratory movement upon the affected side is diminished. The expectoration of the casts is attended with distressing cough and suffocative phenomena. They are usually ejected in a ball-like coil embedded in mucus and blood. Unrolled they appear as large, whitish, arborescent forms, of which the main trunk is sometimes 2 cm. in diameter and as many as 10 cm. in length. On cross section they are solid, sometimes with a minute central canal, circumferentially stratified, containing minute bubbles of air, and, in some cases, little clots of blood. Microscopically there are present red and white blood-corpuscles, alveolar epithelium, and in some cases Charcot–Leyden crystals and Curschmann's spirals. Upon the expectoration of the casts the symptoms are immediately relieved. The form and size of the casts in repeated attacks is often similar. This fact would only justify the conclusion that the same bronchial distribution has been affected when the localization of the physical signs has occurred in the same region of the chest. The cause of the cast formation in limited branches of the bronchia and its recurrence at intervals is unknown.

Prognosis.—In acute bronchitis recovery is the rule, except in debilitated persons and at the extremes of life; in chronic bronchitis the prognosis as to life is favorable, as to recovery unfavorable; in fibrinous bronchitis the prognosis is uncertain, both as to life and recovery.

ii. Bronchiectasis.

Definition.—Dilatation of bronchial tubes. Dilatation of the finer subdivisions is designated bronchiolectasis. Two principal forms of this anatomical condition are recognized—the cylindrical or fusiform, and the saccular or globular. These are sometimes present in the same lung. As a rule there are several dilatations in different portions of both lungs. Occasionally the bronchiectasis is single, especially in chronic bronchitis with emphysema. A form described as bronchiectasis universalis occurs as a congenital condition and is sometimes encountered in chronic interstitial pneumonia; one lung only is affected. The bronchial tubes are represented by a series of dilatations surrounded by dense sclerotic lung tissue. The ordinary forms are common in chronic phthisis affecting the apex, in chronic pleurisy at the base, and in emphysema. They vary greatly in size. The interior of bronchiectatic cavities is lined with a smooth membrane, from which the normal cylindrical epithelium has disappeared. At the dependent parts, as the result of accumulated secretions, there are areas of ulceration. All the layers of the bronchial wall are stretched and atrophied. The contents are often intensely fetid. Bron-

chiolectasis may occur as an acute condition after the febrile infections, or in chronic form in the bronchitis of old persons.

Etiology. — The mechanical factors are twofold: (a) weakening of the tone of the bronchial wall as the result of impaired nutrition, and soakage and dilatation in consequence of the pressure of the contained air in severe cough, together with the weight of accumulated fluid — *pulsion dilatation;* and (b) traction upon the wall of bronchi in the case of pleural adhesions and hyperplasia of the connective-tissue framework of the lung as in pulmonary cirrhosis—*tension dilatation.*

Symptoms.—In large dilatations the cough and expectoration are characteristic. After several hours, usually twenty-four, during which cough has been slight or absent altogether, a violent and prolonged paroxysm will occur with profuse expectoration. The attack commonly comes on in the morning and often follows a change in posture. The expectoration, which varies in daily quantity from 250 to 750 c.c. and may reach a litre, is often brought up in repeated mouthfuls. This phenomenon is due to the fact that the altered mucosa of the cavity does not react to the gradually accumulating secretion. When, by reason of the amount or upon change in position, there is an overflow into the communicating bronchus, the cough reflex is immediately excited and the paroxysm continues until the accumulation is expectorated. The sputum (see page 474). Hæmoptysis is common, usually slight, occasionally severe. Dyspnœa and cyanosis upon exertion are common. Metastatic abscess of the brain and septic phenomena occasionally occur. As a rule, however, the condition runs a chronic course, with clubbing of the fingertips and incurvation of the nails,—drumstick fingers,—and in some cases a fair degree of health.

Physical Signs.—The physical signs in limited bronchiectasis, and in the acute and chronic forms of bronchiectasis, are not characteristic. When the dilatations are sufficiently large and superficially situated they yield upon examination and often in an exquisite manner tympanitic or amphoric resonance, the cracked-pot sound, Wintrich's phenomenon, and an intensification of the vocal fremitus over the affected area. Whispering pectoriloquy may be present. There are circumscribed flattening over the bronchiectatic cavity and diminished respiratory excursus upon the affected side.

Diagnosis.—Direct.—The diagnosis rests upon the history of a chronic pulmonary affection; the signs of a large unilateral cavity, which are gradually effaced as the secretion reaccumulates, and suddenly reappear after several spells of coughing in which large quantities of sputum are ejected; circumscribed flattening; and limited respiratory movement upon the affected side. There are cases in which, with the distinctive cough and expectoration, the dilatation cannot be located by the physical signs or the X-rays, and others in which, with excessive sputum, only diffuse dilatations of moderate size have been found after death.

Differential.—*Vomicæ.*—The anamnesis is suggestive. In favor of the phthisical origin of the cavity are its location in the upper lobe, especially if the opposite apex or the apex of the lower lobe upon the same side shows signs of consolidation, and sputum that is nummular, or, if

fluid, expectorated at relatively short intervals and in smaller amounts, and containing pulmonary elastic fibres. If tubercle bacilli are present the diagnosis of phthisis is positive. The frequent association of phthisical cavities with bronchiectasis must be borne in mind.

Circumscribed pyopneumothorax with bronchopulmonary fistula, pulmonary abscess, pulmonary gangrene, and putrid bronchitis present points of resemblance to bronchiectasis so close that, in some instances, the differential diagnosis may be extremely difficult. The anamnesis is important. In pyopneumothorax there is the history of the initial sudden discharge of considerable quantities of purulent sputum; in abscess and gangrene a history of sudden development and rapid course; in putrid bronchitis a history of chronic bronchitis without special unilateral localization. Bronchiectasis is invariably a secondary affection of gradual development, and more frequently situated centrally than at the periphery of the lung.

Prognosis.—The cavities tend to enlarge. When a single large cavity can be located and treated by drainage and other appropriate surgical measures, the expectation of relief may be entertained.

iii. Tracheobronchial Stenosis.

Definition.—Narrowing of the lumen of the trachea or bronchial tubes.

Etiology.—The lumen of the tracheobronchial tree may be narrowed above the bifurcation by the pressure of goitre, œsophageal or mediastinal tumors, and aortic aneurism; both above and below the bifurcation by enlarged tracheobronchial glands and neoplasms, especially carcinoma; and below it by pericardial effusion and enlarged bronchial glands. It is also narrowed by conditions which arise within the lumen of the trachea or bronchi, among the most important of which are the following: polypi and other new growths; acute œdema of the tracheobronchial mucous membrane, such as results from the inhalation of irritating fumes, inflammatory thickening of the mucous membrane; croupous exudates, as in infralaryngeal diphtheria and in fibrinous bronchitis; cicatrices, especially in syphilis; exuberant granulations from the irritation of a tracheotomy tube and foreign bodies.

Symptoms.—The symptoms differ in intensity, according to the extent of the obstruction and the rapidity with which it comes on. They are more urgent when the stenosis is sudden and when it is tracheal. They consist of inspiratory dyspnœa, suffocative phenomena, anxiety, restlessness, cyanosis, a tense, full, and slow pulse with inspiratory intermission, and, after a time, venous engorgement, dilatation of the right heart, visceral congestions, and diminution or complete suppression of urine.

Physical Signs.—The facies and attitude differ little from those of laryngeal obstruction. There is inspiratory retraction of the supraclavicular and intercostal spaces and of the epigastrium. If the stenosis be tracheal these signs are bilateral; if it involve a main bronchus they are more pronounced upon the affected side. Percussion yields hyperresonance with a slightly tympanitic quality. The absence of dulness excludes all conditions of inflammatory exudate, consolidation of the lung, pleural effusion, or new growth as the cause of the dyspnœa. The vesicular murmur

is enfeebled or quite inaudible; bronchial breathing is absent; coarse dry or moist râles, often heard at a distance, decrease of vocal fremitus, and a faint or whispering voice are important phenomena of stenosis.

Diagnosis.—The differential diagnosis between laryngeal and tracheobronchial stenosis is of the highest practical importance. Among the symptoms which point to the larynx as the site of the obstruction are the abnormally increased respiratory movements of the larynx, the fixed attitude with the head somewhat thrown back, and the peculiar croupy, metallic quality in the respiration and cough—laryngeal cough. The question, however, is immediately settled by an examination with the laryngoscope.

Causal Diagnosis.—The examination of the trachea with the laryngoscope, when practicable, will determine the presence or absence of stenosis in that organ, and, when present, its nature. But there are often difficulties in the examination which are insurmountable. Goitre is manifest; tumor of the œsophagus is associated with dysphagia; mediastinal new growths and aneurism of the arch of the aorta are attended in common by signs of tumor, with displacement of the anterior border of the lung and venous obstruction, and separately by signs which, in the case of aneurism when present, are distinctive, as thrill, diastolic shock, and systolic pulsation. The presence of enlarged bronchial glands may be suspected when tuberculous lymph-nodes are elsewhere present. Malignant disease may be suspected when there are intermittent hemorrhagic sputum, enlarged lymphatics in the neck or axilla, and an otherwise inexplicable cachexia. A history of syphilis and specific lesions of the palate or larynx would lend importance to the assumption of cicatricial stenosis. The recurrence of attacks of fibrinous bronchitis with the expectoration of characteristic casts renders the diagnosis positive. Foreign bodies are easy of diagnosis. There is almost always a history, though under most unusual circumstances or in the case of the insane no history can be obtained. A fixed attitude, sudden attacks of suffocation, and the result of X-ray examination in the case of metallic and certain other substances are of diagnostic importance.

Prognosis.—In tracheobronchial stenosis the prognosis is as variable as the cause.

iv. Bronchial Asthma.

Nervous Asthma.

Definition.—A neurosis characterized by paroxysmal dyspnœa, a sense of constriction of the chest, and irregular recurrence. The symptoms and signs denote hyperæmia and swelling of the mucous membrane of the finer bronchial tubes, and the attack may be produced by direct or reflex irritation.

Asthma is not to be confounded with the dyspnœa of cardiac or renal disease, or that supervening upon exertion in emphysema and other chronic affections of the lungs and pleuræ.

Three principal hypotheses are advanced: (1) that the attack is due to spasm of the bronchial muscles; (2) that it is the result of hyperæmia and turgescence of the bronchial mucosa; (3) that it is caused by a peculiar inflammation of the bronchioles. It is probable that all three

of these conditions—namely, swelling, spasm, and exudate—are present at the same time. Other views of less importance attribute the affection to spasm of the diaphragm or of all the inspiratory muscles. It has been suggested that the condition is similar to that in hay fever, with special manifestations due to differences in the anatomical site of the lesions. The sudden onset of the symptoms, the common association of asthma and hay fever, and the neurotic constitution of the subjects of these affections lend probability to this opinion.

Etiology.—Predisposing Influences.—The neurotic temperament, which is transmitted from generation to generation, frequently carries with it the tendency to asthma. Asthma and epilepsy are sometimes associated. Males are more liable to the affection than females. Age plays an important part in the predisposition. Asthma usually begins early in life, often in childhood, and may continue throughout life. Pertussis is sometimes followed by asthma, and chronic bronchial catarrh is often accompanied by the attacks. These attacks are sharply differentiated from the dyspnœa which attends exertion, from which they are to be distinguished. Idiosyncrasy is important.

The Exciting Cause.—The odors of certain plants or flowers, hay, artificial perfumes, ipecacuanha, and the emanations from animals, as the horse or cat, immediately cause the attack in certain persons. Violent emotions, especially if disagreeable, may act in the same way. Excesses at the table and certain articles of diet may be followed by the outbreak. Many persons remain free from the disease in the city, but at once suffer in the country or in some particular part of the country, or suffer in the city, but miss the attacks in the country or at the sea-shore. Others cannot use a feather pillow or sleep in a particular room. The most common source of reflex irritation is to be found in the mucosa of the upper respiratory tract. Forms of rhinitis, nasal polypi, hypertrophies of the inferior turbinated bones, enlarged tonsils, or adenoid vegetations are frequently present in asthmatics and relief very often follows their proper surgical treatment. The causal influence of uterine and ovarian disease is much less than at one time supposed. In old cases every "cold" may be attended with the paroxysm.

From the standpoint of causation the cases of bronchial asthma may be divided into two groups: First, the anaphylactic or sensitive, in which the attacks begin in early life and are precipitated by the action of various proteins, which serve as asthmogenic substances and are either of bacterial origin, or present in certain articles of food among which are egg albumen, cassin, and fish. The employment of various sera in experimental and human medicine has shown horse serum to belong in the list of these substances. The emanations from the horse and cat not only cause the attack in members of this group but the injections of preparations of the proteins in horse dandruff and the hair of cats also relieve the attacks. Other members of this group of asthmatics are sensitized to the pollen of certain plants as rag-weed, golden rod and timothy, and are relieved by the inoculation of emulsions of these pollens. The part played by bacterial proteins is not so evident. The subject belongs to focal infection.

Streptococcies viridens, or S. hemolyticus or the influenza bacillus may be the local organism. Sensitization from the teeth or the nose—*nasal asthma,* or the genitalia in both sexes—*asthma sexuale* or the upper respiratory tract or bronchi—*asthma bronchialis*—may result. If there is reason to suspect a bacterial cause the form should be sought by every diagnostic means.

The second group comprises those asthmatics who are not sensitized to any of the bacterial or other proteins. In these cases, the attacks first appear in middle life and the subjects suffer from chronic bronchitis, epiphysema or disease of the heart or kidneys. In these cases the dyspnœa which comes on with exertion or attends any exacerbation of the characteristic symptoms of the underlying disease—*cardiac* or *renal dyspnœa*—lacks the special features of asthma and is to be distinguished from it. When improvement in the general symptoms takes place, however, the asthmatic attacks diminish in frequency and severity, though the respiratory difficulties incident to the primary malady continue to occur.

Symptoms.—The health is often excellent in the intervals between the attacks. The onset is sometimes preceded by prodromes, among which are chilliness, oppression in breathing, dyspeptic phenomena, vesical irritability, and mental depression. The attack mostly comes on at night, the patient waking from sleep with distressing difficulty in breathing, and oppression. There rapidly develops a paroxysm of the most urgent dyspnœa. Inspiration and expiration are both affected. The patient struggles for air. The respiratory muscles, and especially those which aid in expiratory efforts, are brought into forcible action. The abdominal muscles are tense and board-like. The expiration is prolonged. The face is pale, the expression anxious, and the patient refuses to talk. He may rush to the open window and gasp for air, with his arms fixed upon the frame. Small quantities of high-colored urine are passed at short intervals. There is a short, dry cough, with a peculiar, scanty, viscid expectoration. The duration of the attack varies from a bad quarter of an hour to half a day or longer. There are cases in which, with remissions and exacerbations, the symptoms last for thirty-six or forty-eight hours. In severe paroxysms air hunger becomes urgent; restlessness, pallor, and cyanosis are accompanied by sweating cold extremities, and a small, quick pulse. The intensity of the symptoms now abates; the cough becomes loose, the expectoration fluid and free, large quantities of urine may be passed, and in a short time there is complete relief. The patient now usually falls asleep. He may awake quite well or one or more further attacks may follow. Urticaria and in rare instances angioneurotic œdema have been observed during the attack. The sputum in the beginning of the attack contains Curschmann's spirals, Charcot-Leyden crystals, together with many leucocytes, mostly eosinophiles.

Physical Signs.—Inspection.—The chest has the inspiratory form. It appears large and suggests emphysema. The condition is, in fact, an acute emphysema, such as occurs also in pertussis. Under these circumstances the residual air is increased and the tidal air diminished, and in proportion as the ratio between them is deranged the chest becomes fixed

and the dyspnœa urgent. The short, quick inspiration and the prolonged expiration are of diagnostic importance. PERCUSSION.—In mild attacks the signs upon percussion are little modified, the change amounting simply to a moderate hyperresonance, but in severe attacks the percussion sound is vesiculotympanitic. The pulmonary resonance extends downward two or three interspaces or more, the superficial cardiac dulness is much diminished, and the margins of the overdistended lungs scarcely change their position with the respiratory movements. AUSCULTATION.—The vesicular murmur is enfeebled. Great numbers of sibilant and sonorous râles are heard in all parts of the chest, and often from every part of the room. These râles constantly change in quality, pitch, and loudness, and are much more prolonged and intense upon inspiration than upon expiration. With free expectoration the râles become moist. The attacks recur at varying intervals. They sometimes come on in a series of three or four at night, with catarrhal symptoms in the daytime.

Diagnosis.—DIRECT.—The clinical picture is distinctive. Among the important criteria are sudden onset, mostly at night; expiratory dyspnœa; acute overdistention of the thorax as shown by the physical signs upon inspection and percussion; scanty expectoration with Curschmann's spirals and Charcot-Leyden crystals; loud wheezing and groaning râles; later abundant expectoration, with moist râles and relief of dyspnœa; eosinophilia. The test for sensitization consists in the intradermic injection of minute amounts of the suspected protein—the so-called skin test.

DIFFERENTIAL.—*Emphysema and Chronic Bronchitis.*—The association of emphysema and bronchial asthma is a double one. The asthmatic tends to become emphysematous on the one hand, while, upon the other, attacks of asthma are common in emphysema. The obvious relationship between these conditions and chronic bronchitis has already been indicated. *Spasm of the Glottis.*—There may be true spasm, as in the laryngeal crisis of tabes. The dyspnœa is inspiratory and noisy, the respiratory movements of the larynx are extensive. The lungs are not overdistended, there is inspiratory retraction of the epigastric zone, and the peculiar cough, râles, and expectoration of asthma are not present. Adductor spasm is of short duration, while the paroxysm of asthma is often prolonged. *Laryngismus Stridulus.*—This form of adductor spasm of children is characterized by apnœa, followed upon relaxation by a long-drawn inspiratory crowing sound. *Cardiac and Renal Dyspnœa.*—So-called cardiac and renal asthma have nothing in common with true asthma except dyspnœa. To call them asthma is a nosological error alike inconvenient to the teacher and misleading to the student. In old asthmatics, after the systematic removal of the usual foci of infection in the nose and accessory sinuses, the tonsils and alveolar-dental structures by operation, the chronic bronchitis commonly present may supply the bacterial protein which causes the attacks. It is in the highest degree probable that such an autogenous source of protein poison may be active in certain cases of asthma. No effort should be spared to localize and eradicate the *gens et virgo mali* in any severe case. The cases of obscure source, perhaps for the very reason that they are obscure, are the most refractory to treatment.

Complement-fixation and precipitin reactions, using the patient's serum

and various proteins, were found to be of no help in diagnosis or prognosis or treatment of the condition.[1]

Prognosis.—The symptoms are often alarming, but death does not occur during the attack. The removal of the sources of reflex irritation or protein infection in the upper air-passages or elsewhere, improvement in the general condition of the patient, or permanent residence in a suitable climate is often followed by lasting relief. Many of the cases are, however, incurable. The tendency in asthmatics is to the development of chronic bronchitis and the emphysematous chest. There is always the danger of croupous or bronchopneumonia. Nevertheless many asthmatics reach advanced years.

IV. DISEASES OF THE PULMONARY TISSUE.

i. Circulatory Derangements.

(a) **Pulmonary Congestion.** — Congestion of the lungs is usually a symptomatic affection. There are two forms — active and passive.

1. **Active Congestion of the Lungs.** — The inhalation of overheated air, smoke, and other irritating substances, and overaction of the heart may cause this condition. The sudden death of firemen, open-air orators, and drunkards after exposure has been ascribed to it. The symptoms comprise great dyspnœa, oppression, feeble pulse, and cyanosis. The physical signs are restricted respiratory movements, impaired resonance, faint vesicular murmur, and fine râles. The mechanical interference with the circulation in pneumonia, intense bronchitis, pleurisy, and tuberculosis leads to overdistention of the capillaries in the adjacent lung tissue—collateral fluxion. The importance of this condition arises chiefly from the danger of œdema.

2. **Passive Congestion.**—Two forms are recognized—mechanical and hypostatic. Mechanical Congestion.—The condition is most marked in the dependent portion of the lungs. The essential factor is an obstacle to the return of the blood to the left ventricle. Its occurrence is favored by all conditions which restrict the respiratory expansion and contraction of the lungs and thus interfere with the normal movement of the blood current in the pulmonary vessels. Mechanical congestion of the lungs occurs in mitral stenosis and incompetency, emphysema, and in consequence of the pressure of tumors. The lung undergoes the changes known as brown induration. So long as compensation is maintained this condition is not marked by special symptoms of importance. When it is lost, dyspnœa, cough, and expectoration, often blood-stained and containing alveolar cells with blood pigment, occur. Hypostatic Congestion.—The bases in this condition also are engorged with blood and serum. The condition is bilateral, one side being usually more deeply and more extensively congested than the other. Lobular patches may be airless, and bits of the affected tissue may sink in water. To this extreme condition are applied the terms splenization and hypostatic pneumonia. In fact there are frequently present in the congested regions foci of bronchopneumonia. Hypostatic congestion is common in protracted acute illness, as enteric fever; in chronic wast-

[1] I. C. Walker, Ins. Amer. Med. Association, Aug. 4, 1917.

ing diseases, as tuberculosis and cancer; in injury and disease of the brain, especially apoplexy, and in prolonged coma. There are no characteristic symptoms and the diagnosis rests upon the presence, over the lower lobes posteriorly, of impaired resonance, feeble respiratory sounds, patches of bronchovesicular breathing, and small mucous or subcrepitant râles.

(b) **Pulmonary Œdema.**—There are two forms of œdema of the lungs—general and collateral.

The termination of intense congestion of the lungs is, in many cases, the transudation of blood-serum from the overdistended capillary vessels into the vesicles themselves and their walls. The escape of serum into the small and later into larger bronchi follows and is of clinical importance. Pathologically the condition is one of serous infiltration of the pulmonary tissue, with accumulation in the air-cells and bronchi. The œdematous lung is heavy, pits on pressure, and exudes abundantly from the cut surface clear or blood-tinged serum.

1. **General Œdema.**—*Œdema from Engorgement; Stasis Œdema.*—The œdema is bilateral and involves the whole of both lungs. The bases are especially affected. Causal factors are overdistention of the capillary vessels, hydræmia which leads to nutritive changes in the walls of the vessels and a weakened left ventricle. The condition is very often a terminal one and accompanies the death agony. It is common in affections characterized by dropsies, as fatal anæmias, disease of the heart and kidneys, especially the cardiorenal affection, and cachexias generally. It occurs also without previous dropsy in cerebral diseases, acute pulmonary congestion, and angina pectoris. General œdema of the uninvolved portions of the lungs may occur in the *stadium ultimum* of croupous pneumonia.

2. **Collateral Œdema.**—*Local Œdema of the Lungs.*—This condition is the outcome of the collateral fluxion in the pulmonary tissue bordering on pneumonias, infarcts, active foci of tuberculous inflammation, or new growths. The cut surface exudes a bloody serum. The entire lung is not involved and the opposite lung may wholly escape. This constitutes the form known as inflammatory œdema.

Symptoms.—Pulmonary œdema may develop gradually or with great suddenness. The symptoms of the pre-existing malady are aggravated. Progressive dyspnœa, cough, copious, frothy, thin, fluid sputum which, in the case of collateral œdema, is often bloody, characterize the condition (see Part III, page 474). As it progresses cyanosis and the stupor and convulsive tremblings which indicate the action of carbon dioxide upon the nervous system occur. Fever does not usually accompany stasis œdema, but in inflammatory œdema there may be a rise in temperature.

Physical Signs.—The percussion resonance is usually somewhat impaired over the bases posteriorly and has the tympanitic quality. In very abundant serous transudation there may be dulness. Upon auscultation the respiratory murmur is enfeebled, and over the whole extent of the involved lung tissue are heard moist bronchial subcrepitant and crepitant râles. Vesiculobronchial or pure bronchial breathing may be heard in limited areas at the bases in intense œdema, and corresponds to the areas of dulness.

Diagnosis.—DIRECT.—The diagnosis of general œdema of the lungs rests upon the occurrence of the above described symptoms in cases of œdematous or cachectic disease, cerebral disease or injury, angina pectoris, and impairment of the power of the left ventricle, especially when the power of the right heart is fairly well maintained. Collateral œdema may be at least provisionally diagnosticated when, in pneumonia or other inflammatory conditions, infarct, active circumscribed tuberculosis, or new growths, the symptoms are aggravated, the temperature rises, many moist râles are heard in the adjacent lung, and there is an abundant thin, blood-stained sputum. In rare instances acute œdema of the lungs follows the withdrawal of a pleural effusion by aspiration. The sputum is copious and has the characters above described. It is the result of the sudden removal of pressure upon the pulmonary vessels. Even more rare is the perforation of the lung by a serous pleural effusion. There are coarse râles usually confined to the affected side, and an abundant expectoration presenting the characters of the sputum in œdema but with a larger albumin content.

Prognosis.—General œdema of the lungs is frequently one of the manifestations of dissolution. There are, however, cases that recover under proper treatment. The outlook is at the best uncertain. Collateral œdema may mark an unfavorable turn in an acute illness or, as is frequently the case, subside under energetic management and be the point of departure for lasting improvement.

(c) **Pulmonary Hemorrhage.**—There are two forms. In the first the blood escapes into the bronchi and is expectorated—bronchopulmonary hemorrhage; in the second the blood is effused into the tissue of the lungs and air-cells—pulmonary apoplexy, hemorrhagic infarct.

1. **Bronchopulmonary Hemorrhage.**—*Bronchorrhagia; Hæmoptysis.* (See Part III, page 474.)

2. **Pulmonary Apoplexy.**—*Pneumorrhagia; Infarct.*—Anatomically two conditions are encountered: diffuse infiltration and hemorrhagic infarct.

Diffuse Hemorrhagic Infiltration of the Lungs.—The lung tissue and air-cells are densely and uniformly infiltrated with extravasated blood. The cut surface presents a smooth, somewhat gelatinous appearance and a blackish color. The condition is rare. It occurs more frequently in the hemorrhagic fevers, less often in sepsis and acute cerebral disease. The symptoms are dyspnœa, cyanosis, bloody sputum, blackish in color, and the nervous phenomena of collapse. Resonance is impaired. This form of hemorrhage is of no great diagnostic importance, since it constitutes the terminal event in an otherwise fatal malady.

Hemorrhagic Infarct.—The extravasation of blood is due to the arrest of circulation in a branch of the pulmonary artery by an embolus or thrombus. The anatomical condition and the symptoms differ greatly according to the location of the occlusion. If it occurs in the trunk or a main branch of the pulmonary artery, the whole or a large part of the blood is prevented from entering the pulmonary circuit, and there is dilatation of the right heart, a small, thready, arterial pulse, intense dyspnœa, cyanosis, and death from apnœa—*pulmonary apoplexy.* As the clinical manifestations from cardiac paralysis are the same the diagnosis remains an uncertain one

When the obstruction takes place in a smaller branch of the pulmonary artery infarction usually occurs. These lesions are commonly at the periphery of the lung, and wedge-shaped, with the base resting upon the pleura, which is inflamed. Exceptionally they are located within the tissue of the lung and they are then irregularly oblong. Recent infarcts present the appearance of a blood-clot in the pulmonary tissue. The air-cells and their walls and the capillaries are packed with red blood-corpuscles. Infarcts are commonly multiple, exceptionally single. They vary in size from a pigeon's egg upward and may occupy a large portion of a lobe. In the arterial branch of supply may commonly be found the embolus or thrombus in the neighborhood of the apex. These obstructions, notwithstanding the fact that the pulmonary arteries are terminal, do not always cause infarction, owing to the width and free anastomosis of the capillaries and the ability of the bronchial vessels to maintain the circulation. The changes in the infarct are similar to those in blood-clots in other situations. The color becomes reddish-brown; the tissues contract, and are finally converted into a puckered, pigmented, fibroid nodule. The source of the embolus is to be sought in the right heart or peripheral venous system. The white thrombi which form in the right auricular appendix, the vegetations which develop upon the tricuspid leaflets in the rare cases of right-sided endocarditis, fibrin formations among the columnæ carneæ may be swept by the venous blood stream into the ramification of the pulmonary artery and become lodged. Any condition which tends to weaken the action of the right ventricle predisposes to this accident. Among these are valvular disease, especially mitral affections and myocardial degenerations. These emboli are not usually septic. Emboli from inflammatory or suppurating foci in various regions are infected and cause not a simple infarct but a metastatic abscess. When the general condition is septic, numerous small suppurating foci develop in the lung—*pyæmic abscesses*. These cannot always be recognized during life. When, however, a more extensive portion of the lung undergoes septic infarction pulmonary abscess results.

Symptoms.—The symptoms are neither constant nor distinctive. An initial chill may occur. It is, however, never so severe or prolonged as the ordinary chill of croupous pneumonia. Cyanosis, increased respiratory frequency, and dyspnœa at once develop. These symptoms vary in proportion to the number, and especially the size, of the infarctions. They may be slight or altogether absent. There is cough and the sputum contains blood (see Part III, page 475).

Physical Signs.—There is circumscribed dulness, more commonly in the lower lobes, especially on the right side, with bronchial breathing and high-pitched small mucous râles. Pleural friction over a limited area may very often be demonstrated.

Diagnosis.—DIRECT.—The diagnosis cannot always be made with certainty. The sudden occurrence of the above rational symptoms and physical signs in the course of chronic disease of the heart, or thrombosis of a crural or other vein, or some distant inflammatory or suppurative process warrants a provisional diagnosis.

Differential.—*Croupous Pneumonia.*—There is a superficial resemblance in some of the cases; but the situation and outline of the consolidation, the character of the sputum in which the blood or hæmoglobin is more intimately mixed than in infarction, and the results of the laboratory examination of the sputum, which contains pneumococci, are distinctive. *Hæmoptysis in Mitral Disease, especially Mitral Stenosis.*—The occurrence of blood spitting, usually in small amounts and extending over a period of days or weeks, may suggest infarction. The differential diagnosis in the absence of physical signs or marked pulmonary symptoms is impossible. The blood may be due to engorgement of the pulmonary vessels. Its recurrence after exertion and at long intervals is in favor of the latter view. *Malignant Disease of the Lung.*—Blood spitting and signs of consolidation are present in both conditions. Pain, wasting, localizing physical signs, which gradually include more territory, cachexia, the presence of new growths elsewhere, and in particular of pigmented nævi or warts, with secondary nodules in the skin or subcutaneous tissues, implication of the lymph-nodes, or a history of the removal of a malignant growth, justify a provisional, and in well-marked cases a positive, diagnosis of cancer or sarcoma of the lung.

Air Embolism; Fat Embolism.—An embolus is any body transported by the circulating blood and capable, when arrested by the narrowness of the vessel, of obstructing the circulation. Emboli are usually too large to pass through the capillaries. They may be composed of fibrin masses, fragments of thrombi, vegetations or calcareous particles from endocardial vegetations, or fragments of neoplasms, which have penetrated the wall of a vessel. They may be infected or non-infected. There are two substances, differing from ordinary emboli in not consisting of solid bodies, which may give rise to urgent or fatal consequences when arrested in the capillaries of the pulmonary circulation; these are *air* and *fat.*

Etiology.—Air embolism occurs under certain circumstances when a vein is lacerated. Fat or oil embolism may follow fracture or injury to a bone, with escape of marrow into the tissues, or extensive laceration of adipose tissue, or its rapid breaking down in suppurative processes.

Symptoms.—The symptoms of air embolism are urgent or even fatal in cases in which the quantity of air is sufficient to form large numbers of bubbles which cannot pass the pulmonary capillaries—*an embolic shower.* They consist of an extreme degree of air hunger, loss of consciousness, convulsions, and collapse, and usually prove fatal in a brief period, sometimes instantly. In rare cases, however, these most alarming manifestations improve in consequence of the rapid absorption of the air, and prompt recovery takes place—a fortunate event not seen when multiple embolism, the embolic shower, is due to solid emboli. When the air bubble entering the vein is small or the air enters slowly the symptoms are commonly less urgent. No symptoms attend the presence of the most minute air bubbles which, having passed the wider pulmonary capillaries, are arrested in the capillaries of other organs.

Since the fat globules obstruct only the finest vessels in the lungs and only gradually enter the circulation, the sudden pulmonary symptoms seen in air embolism rarely present themselves. As a rule no symptoms

occur until some hours or days have elapsed from the time of the injury. Severe dyspnœa, œdema of the lungs, great depression, and coma may occur. As the fat emboli have no tendency to cause blood coagulation or thrombosis, they are gradually forced on under the pressure of the blood stream, or undergo resorption, and recovery takes place. The fat particles which pass the lungs may reach the cerebral capillaries or be arrested in the renal glomeruli. In the latter case they may be voided in the urine. Death is uncommon in fat embolism.

ii. Diseases Characterized by Changes in the Vesicular Structure of the Lungs.

(a) PULMONARY EMPHYSEMA.

Vesicular Emphysema; Substantive Emphysema; Pseudohypertrophic Emphysema.

Definition.—A chronic disease of the lungs, in which the infundibula and vesicles are dilated and their walls atrophied.

This is a well-defined clinical affection, and characterized by enlargement of the lungs, changes in the contour of the chest, incomplete aeration of the blood, and varying degrees of dyspnœa, especially upon exertion. It is to be distinguished from acute vesicular emphysema, compensatory emphysema, and interstitial and atrophic forms.

Etiology.—Heredity constitutes an important predisposing influence. The disease is frequently encountered in successive generations or in several members of a family, and is not uncommon in childhood. It has been ascribed to congenital defects in the development of the elastic fibres. Long-continued habitual intra-alveolar pressure, acting upon a congenitally defective alveolar structure, causes distention which tends to become permanent. The hyperinflation of the lungs which occurs in the paroxysms of whooping-cough and asthma is often the starting-point of emphysema. Repeated attacks of bronchitis or chronic bronchitis are often present. It is common also in players upon wind instruments, glass-blowers, and those whose work demands heavy lifting or prolonged muscular strain. The tension under these circumstances is expiratory. In violent attacks of cough and in straining, the glottis is closed and the intrathoracic tension greatly heightened. The parts of the lungs least supported by the chest wall, namely, the apices and anterior and inferior margins, show the most developed lesions of emphysema.

Anatomically the primary changes are in the lungs; the secondary changes in the wall of the thorax.

The lungs are voluminous, their margins meeting in the anterior mediastinum and extending downward to the extent of two fingers' breadth or more. The diaphragm is correspondingly displaced in a downward direction. They have lost their normal contractility and do not retract when the costal pleura is incised nor when withdrawn from the chest and laid upon the table. At the apices and borders there are seen beneath the pleura greatly distended air-vesicles, varying in diameter from

1 to 3 mm., and sometimes attaining the size of a pigeon's egg. There is marked diminution in the pigment usually found in the subpleural lymph-spaces. The atrophy of the distended vesicular walls leads to loss of their pumping function and permanent pressure upon the capillaries, and this to diminution in the intervesicular vascular supply. The infundibula are dilated, but bronchiectasis is not very common. The chambers of the right heart are dilated and hypertrophied. The pulmonary artery is in some instances dilated and atheromatous.

The chest permanently assumes the inspiratory form and the costal cartilages progressively undergo calcification and lose their elasticity.

Symptoms. — The derangement of function is twofold. First, the residual air is greatly increased and the tidal air correspondingly decreased in volume, and second, the pulmonary circulation is diminished. If these facts are borne in mind the significance of the symptoms is obvious. The lesions are gradually developed and it is only after they have made some progress that the characteristic symptoms and signs occur. At first there is merely dyspnœa and faint lividity upon exertion, and the chest merely looks full with the inspiratory contour. When the disease is fully developed the following symptoms are present: *Dyspnœa.*—The elasticity of the vesicular structure being to a great extent impaired, expiration is prolonged and difficult. The dyspnœa is chiefly expiratory. The loss of resiliency in the costal cartilages, the permanent maximum distention of the thorax, and the restricted play of the diaphragm render inspiration also difficult, even with the aid of the auxiliary muscles of respiration. The dyspnœa may be felt upon slight exertion or it may be continuous. It is increased upon the occurrence of the exacerbations of bronchitis, to which the patient suffering from emphysema is so liable. The breathing is puffy and wheezy. *Asthmatic Attacks.*—The dyspnœa under certain circumstances, and especially after "taking cold," often assumes a paroxysmal intensity, differing in no respect from true spasmodic asthma. *Cough* is a common symptom. It is due to the associated bronchitis. It is commonly wheezy and feebly explosive, and without much expectoration. It is usually less troublesome in warm, dry weather, and constitutes the recurrent winter cough of many elderly persons. *Cyanosis.*—The patients are frequently able to go about with lividity of high grade. This symptom is variable and may amount merely to a certain blueness of the lips and finger-nails while the patient is at rest.

Intermittent Cervical Hernia of the Lung of Spontaneous Origin.—C. B. Farr has reported a case of this kind and collected seven other instances from the literature. As a rule the condition accompanied chronic bronchitis with emphysema. It was unilateral in five cases and bilateral in three. These protrusions are of the form and size of a pear, with the base below and the apex extending upward. They are not present upon quiet breathing but prominent during cough and are the seat of a faint rustling murmur. They are resonant on percussion and may be reduced by gentle taxis. In several of the cases a hernial ring could be felt. They are to be differentiated from enlargements of the sinuses of the jugular veins, abscesses, and an empyema which presents in the neck.

Physical Signs.—*Inspection.*—In advanced cases the deformity of the chest is typical. It is barrel-shaped (see p. 64, Vol. I). The elevation of the sternum and ribs gives the neck a shortened appearance. The respiratory movements appear forcible, but the thorax does not expand. Many dilated superficial venules are seen along the line of attachment of the diaphragm. The cardiac impulse is not visible. Epigastric pulsation and dilated cervical veins, sometimes pulsating, are signs of a dilated and overdistended right heart. The deformity of the chest is less marked in those cases in which emphysema has commenced in advanced life at a period when the cartilages have already become calcified. *Palpation.*—The lack of respiratory excursus is very obvious upon palpation. The vocal fremitus is enfeebled. The impulse of the heart cannot be located. There is pulsation over the lower sternal and epigastric regions. *Mensuration.*—The restricted expansion of the chest is confirmed by careful measurement, and the rounded contour by the cyrtometer. *Percussion.*—The resonance is of vesiculotympanitic quality, especially sonorous over the lateral and posterior regions. The percussion sound has been compared with that elicited upon tapping a bandbox—*Schachtelton.* More important than the quality of the percussion sound are the borders of the expanded lungs which it marks. They are extended in every direction and may reach in front to the eighth rib and below it, behind to the level of the twelfth dorsal or even the second lumbar vertebra. The cardiac dulness may be completely obliterated. The liver and splenic dulness are much lowered. *Auscultation.*—The vesicular murmur is greatly enfeebled. Its very faintness is suggestive. The expiratory element is not often audible. Instead of it, however, there are many sibilant and wheezy râles. The sounds of the heart are faint and distant, the pulmonary second sound is often accentuated, and in advanced cases a tricuspid regurgitant murmur may be heard.

The effect of the lesions of emphysema upon the circulation is to obstruct the pulmonary circuit; to dilate and enfeeble the right ventricle; to diminish the arterial blood; and to increase the accumulation of venous blood. The enfeeblement of the right heart is, however, delayed by compensatory hypertrophy by which the circulatory faults are postponed. Ultimately, however, compensation fails and the results of venous engorgement become manifest. These are persistent cyanosis, pulsation in the veins of the neck, hepatic enlargement, diminished secretion of albuminous urine, œdema, anasarca and effusions into the great serous sacs, and gastric and intestinal catarrh. When these conditions are present there is almost always a complicating catarrhal bronchitis, manifest by the ordinary symptoms of cough and expectoration, the latter being usually mucoid and viscid, sometimes purulent. The presence of blood is not common and suggests either a complicating tuberculosis or pulmonary infarct. The general nutrition is impaired.

Diagnosis.—Direct.—In well-developed cases the condition may be at once recognized by the contour and diminished mobility of the chest, the dyspnœa and cyanotic lips, the spare frame, and a facies which is very suggestive to those who have observed many cases. The physical signs, and especially the displaced boundaries of the lungs as determined by percussion, are confirmatory.

Differential. — *Acute Vesicular Emphysema; Acute Over-inflation of the Lungs.*—During the paroxysm of bronchial asthma and pertussis, and in bronchitis of the smaller tubes, the lungs are frequently much distended. The chest maintains the inspiratory form, and upon a single examination the condition might be confounded with emphysema. The anamnesis is important. The borders of the lung are enlarged, but not beyond the limits of normal full-held inspiration, and in the course of a little time after the termination of the primary disease they regain their normal position upon inspiration and expiration. In congenital weakness of the lung tissue such attacks may become the point of departure for true emphysema. A similar condition occurs in cases of cardiac dyspnœa and angina pectoris. The lungs are distended, their borders extended, and the expiration is prolonged and accompanied by wheezing râles. *Compensatory Emphysema; Vicarious Emphysema.*—When local lesions in the lung or pleura interfere with expansion upon inspiration, the unaffected tissue takes upon itself increased functional activity—*vicarious respiration.* This change may involve parts adjacent to the lesion, an adjoining lobe, or the opposite lung. It occurs in bronchopneumonia and around tuberculous foci and cicatrices, the air-cells of the unaffected lobules undergoing a vicarious distention; in pulmonary cirrhosis, the unaffected lung undergoing vicarious enlargement, and to a less extent in pleural adhesions and effusions and in pneumothorax. This process, at first truly compensatory and physiological, becomes after a time pathological. The vesicular walls and capillaries undergo atrophy; a circumscribed or partial emphysema comes to pass. In bronchopneumonia or pulmonary tuberculosis with scattered lobular lesions the distention of the adjacent air-cells masks the dulness and may render the diagnosis obscure; in retracted and cirrhotic conditions of one lung, such as follow the resorption of the pleural effusion, the borders of the opposite lung are much distended and may be followed by careful percussion, not only in their inferior extent, but also over the area of superficial cardiac dulness and beyond the median line toward the contracted side. *Atrophic Emphysema; Atrophy of the Lungs; Senile Emphysema.*—This is a purely senile change and is occasionally encountered in wizened old people with small, narrow chests which are permanently in the expiratory form. The alveolar walls and interalveolar capillaries are extensively atrophied, and the alveoli in places converted into series of large communicating cells. The lungs themselves are small and the thorax conforms to the changes in the contained organs. It is flattened, the shoulders droop, the costal angle is acute, the neck appears elongated. The diaphragm is high. The right heart does not show dilatation and hypertrophy as in large-lunged emphysema, because it also undergoes a corresponding senile involution. The respiratory muscles are atrophic. *Interstitial Emphysema.* — Small bubbles of air find their way into the interlobular and subpleural tissues. Their access is by way of tracheotomy or other surgical or accidental wounds of the neck or throat; less frequently through rupture of the alveolar walls by violent coughing, contusions of the chest, or ulceration. When the opening is near the root of the lung air may pass to the mediastinal connective tissue. The condition is rare. Mediastinal emphysema may be diagnosti-

cated when the causal factors are present together with crepitating subcutaneous emphysema of the neck or chest, absence of cardiac dulness and impulse, obliteration of the sternal ends of the intercostal spaces, and upon auscultation a fine crepitus synchronous with the action of the heart. The veins of the neck are distended. Subpleural and interlobular emphysema not extending to the mediastinum does not usually present positive diagnostic phenomena. Rupture of an air bleb in subpleural emphysema may cause pneumothorax.

Prognosis.—Substantive emphysema is incurable, but much can be done to relieve the sufferings of the patient and to prolong his life. Treatment of the bronchitis, a favorable climate, and attention to the state of the heart are important. Death usually occurs in consequence of some intercurrent disease, as pneumonia or bronchopneumonia.

(b) PULMONARY ATELECTASIS.

Collapse of the Lung.

Definition.—An airless condition of lobules or parts of the lung, the vesicles being in a state of collapse and not occupied by fluid or solid exudate or other pathological products. It is congenital or acquired.

Congenital Atelectasis.—The lung is airless at birth, or remains only partially expanded in consequence of deficient inspiratory efforts or obstruction of the respiratory passages by meconium or mucus. The respiration is feeble, ultimately gasping, shallow and rapid, the lower part of the thorax and the epigastrium are retracted during inspiration, there is universal deep, bluish-black cyanosis, together with muscular twitchings or general shivering convulsions.

Acquired Atelectasis.—This is a secondary anatomical lesion. It is caused in two ways: (a) by obstruction of small bronchi by mucus or swelling of the mucosa—as in bronchopneumonia, the intra-alveolar air undergoing resorption by the capillaries and the vesicles collapsing by virtue of the elasticity of their walls; or (b) by pressure upon the lung such as occurs in pleural or pericardial effusions, pneumothorax and pneumopericardium, tumors of the lungs or pleura, mediastinal tumors or massive enlargement of the heart, scoliosis, and abdominal disorders which restrict the movements of the diaphragm, including persistent meteorism, large ascites, and visceral and other tumors. Diffuse atelectasis may arise in paretic conditions involving the muscles of respiration.

Symptoms.—The clinical phenomena vary according to the extent of lung tissue involved. In slight cases they are not distinctive. There may be moderate dyspnœa, cyanosis, a vesiculotympanitic percussion sound, and enfeebled vesicular murmur. When extensive atelectasis is present, especially if superficial, there are signs of consolidation, namely, dulness, increased vocal fremitus, bronchial respiration, and bronchophony. If there are in the affected region lobules which are incompletely collapsed, the percussion sound has the tympanitic quality and there is persistent fine crepitus. The clinical picture is usually, however, dominated by the symptoms of the primary affection. A condition showing the above symptom-complex which is transitory, that is, which passes off in the course of twenty-four hours, is from this very fact almost sure to be due to atelectasis.

(c) BRONCHOPNEUMONIA.

Lobular Pneumonia; Catarrhal Pneumonia; So-called Capillary Bronchitis.

Definition.—Inflammation of capillary or terminal bronchi and the air-vesicles which constitute the corresponding pulmonary lobules. There are several forms, all of which are caused by bacterial invasion of the lungs. The disease may be primary or secondary and is characterized clinically by symptoms of infection and interference with the respiratory function.

Etiology.—Predisposing Influences.—Bronchopneumonia is common at the extremes of life. In children the relatively small size of the bronchi, their more abundant vascular supply, and the more rapid and exuberant growth of the epithelium of the bronchial mucosa constitute predisposing factors of great importance; while in the aged tissue relaxation, tendencies to passive congestion, and diminished reflex excitability on the part of the bronchial mucous membrane act in the same way. Bronchopneumonia is more prevalent in the winter and spring than at other seasons of the year. It is more common among the poorer classes. In the great majority of the cases bronchopneumonia occurs as a secondary or intercurrent process. There are two principal groups of cases: 1. Those in which it arises in the course of simple bronchitis or an acute infectious disease in which bronchitis forms part of the symptom-complex, as measles, pertussis, diphtheria, scarlet fever, influenza, and less frequently the variolous diseases, erysipelas, and enteric fever. It is a common complication of the acute intestinal diseases of infancy. Bronchopneumonia is a grave complication in all these affections and constitutes the cause of death in the majority of fatal cases. Its frequency corresponds closely to their epidemic prevalence. It is far less common in the acute febrile diseases of middle life. In the aged it is a common complication and frequently forms the terminal event in various acute and chronic diseases. The consolidating lesions of pulmonary tuberculosis are due to chronic localized tuberculous bronchopneumonia. 2. Aspiration or deglutition pneumonia. In the stupor of the low fevers and in comatose states of all kinds the reflex excitability of the larynx is lowered, and the secretions of the mouth, minute portions of drink, and particles of food during the act of swallowing are drawn into the trachea and bronchi. In this situation they set up an active bronchitis which by extension rapidly involves the smaller bronchi and gives rise to an intense bronchopneumonia. This accident is very common after operations upon the throat, nose, larynx, and trachea. Most cases of ether pneumonia arise in this way. The infecting material may come from within the chest itself; severe bronchopneumonia not rarely follows hæmoptysis and occasionally the aspiration of the contents of a bronchiectatic cavity, or pus from an empyema which has found its way into the lung by way of a bronchopulmonary fistula is followed by this disease. The extension of tuberculosis from one part of a lung to another, or to the opposite lung, is due in some instances to aspiration.

Exciting Cause.—Various micro-organisms are associated with the lesions of bronchopneumonia. In the primary form which attacks young children in previous health the common organism is the pneumococcus.

which may be found in pure culture. In the secondary forms the streptococcus is the common infecting agent, but mixed infections are usual. The organisms present are the Bacillus pneumoniæ of Friedländer, Streptococcus pyogenes, Staphylococcus albus et aureus. The Klebs-Löffler bacillus is frequently found in the bronchopneumonia of diphtheria, and Pfeiffer's bacillus in influenza pneumonias.

The lesions consist in interstitial inflammation of the bronchi and alveolar walls. The small bronchi are plugged with exudate composed of leucocytes and swollen epithelium; their walls are swollen, infiltrated with cells, and traversed by distended capillaries, and there is dense peribronchial infiltration. The vesicles are filled with leucocytes and swollen epithelium, and rarely show the dense accumulations of red corpuscles and the fibrillated fibrin seen in croupous pneumonia.

The pathological unit is the inflamed pulmonary lobule. Hence the descriptive term lobular pneumonia. According to the distribution of the lesions three anatomical forms exist: 1. There is more or less intense bronchitis extending to the finer tubes, without the gross evidences of lobular consolidation, but with the microscopic findings of inflammation. This form is bilateral. 2. The foci of inflammation are scattered throughout the lung tissue, with lobular collapse and infiltrated lobules felt as hard nodules. These patches of bronchopneumonia are sometimes isolated with strands of congested or uninflamed tissue intervening and areas of collateral emphysema, and sometimes massed in groups of considerable size. This form is also bilateral. 3. The greater part of a lobe is often involved—the pseudolobar form of bronchopneumonia. Even in this form the consolidation is not uniform as in croupous pneumonia, but there are more or less extensive tracts of deeply congested but still crepitant tissue scattered among the inflamed lobules.

The terminations are in resolution, suppuration or gangrene, or fibrosis. In the tuberculous forms, which are very common in previously apparently healthy children, bronchopneumonia terminates in caseation or chronic fibroid changes. Such cases often follow measles, pertussis, or diphtheria and may be the result of the lighting up of a latent tuberculosis or of tuberculous infection at the time.

Symptoms.—The onset of the primary form is abrupt. It is marked by convulsions and sudden rise of temperature. Cerebral symptoms are frequent and often intense. The defervescence may be critical. These cases, which run a course analogous to that of croupous pneumonia and present similar symptoms and localized physical signs, when they terminate fatally often show pseudolobar consolidation and the pneumococcus in the lesions.

The secondary forms are preceded by the signs of a bronchitis extending to the smaller tubes. The disease develops gradually without chill or convulsion. The fever is of variable intensity—102°–104° F. (39°–40° C.)—and does not conform to type, being irregularly remittent. The skin is hot and dry. There is cough, which is hard, dry, and distressing. The respiration frequency may reach 60 to 80 per minute, and cyanosis soon appears. Other evidences of deficient oxygenation of the blood are frequent, small, thready pulse, stupor, restlessness, and occasional convulsive

tremor. The cough becomes less urgent, the child no longer struggles for air, the face becomes suffused and loses its anxious expression, and death occurs from cardiac paralysis. Sputum (see Part III, page 473).

Physical Signs.—Upon *inspection* there are early signs of obstruction in the terminal bronchi, inspiratory retraction of the intercostal spaces and lower sternal and epigastric regions, rapid, shallow, jerky respiration, and cyanosis of the lips and finger-tips. *Percussion.*—Death may occur before signs of consolidation develop. There may be merely vesiculo-tympanitic resonance. If consolidation is present impaired resonance, sometimes actual dulness, is found at the bases in scattered areas. To

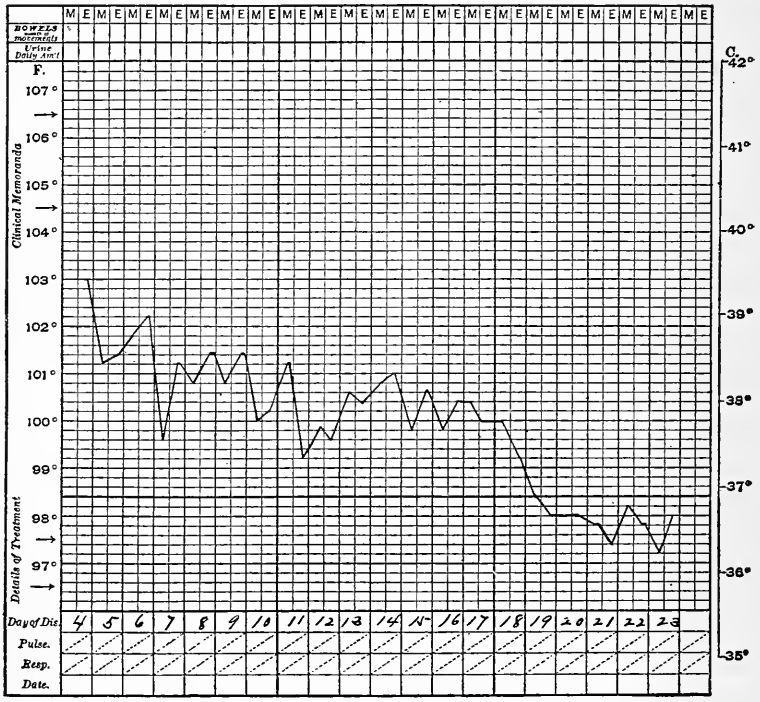


FIG. 318.—Bronchopneumonia in a man aged seventy-six.

elicit it in the latter case demands very careful light percussion. Compensatory emphysema may mask small areas of dulness. *Auscultation* in the early stages reveals only the signs of the extension of the bronchitis to the finer tubes, namely, many diffuse small mucous and some crepitant râles. These are usually more intense and numerous at the bases posteriorly. Vesiculobronchial and, here and there, pure bronchial respiration may be heard over the patches of dulness. Failure of the right heart is followed by pulmonary œdema, which is usually the forerunner of the death agony.

Clinical Varieties. — 1. PRIMARY FORM. — It is a question whether many of these cases are not in truth irregular forms of croupous pneumonia—pneumococcus pneumonia. The primary form is rare in adults.

2. Masked Forms.—The actual condition may in infants be masked by cerebral symptoms such as also occur in croupous pneumonia at this period of life, namely, convulsions, drowsiness, retraction of the muscles of the back of the neck, and stupor; or by gastro-intestinal symptoms, such as nausea, vomiting, and looseness of the bowels. 3. Suffocative Catarrh.—The overwhelming cases were so designated by the earlier writers. The ordinary acute cases of this group, fatal in the course of two or three days, are most appropriately described under this term. 4. Secondary Forms.—Mild secondary bronchopneumonia may follow

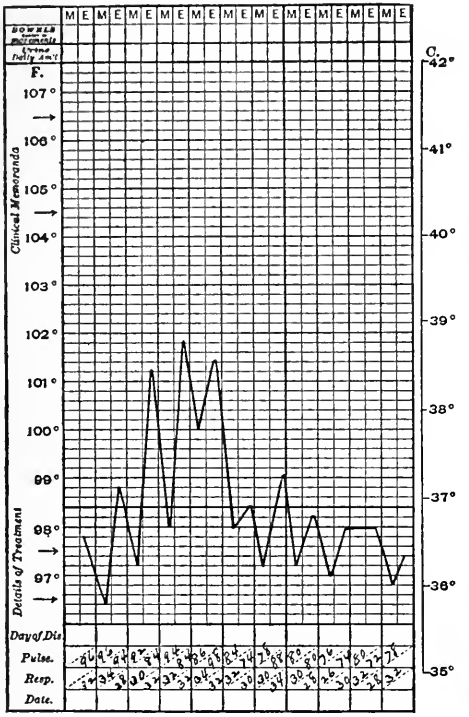


Fig. 319a.—Bronchopneumonia occurring in the course of chronic nephritis.

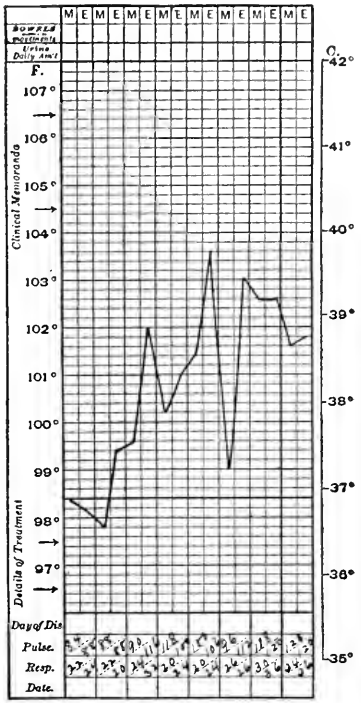


Fig. 319b.—Fatal bronchopneumonia. Man aged sixty-seven.

severe forms of bronchitis, both in children and adults. This form assumes great importance in diagnosis, since it may be merely a simple pathological condition, on the one hand, or an insidiously developing tuberculous bronchopneumonia, upon the other. 5. Ether Pneumonia; Postoperative Pneumonia.—This form of bronchopneumonia usually is pseudolobar and presents close resemblances to croupous pneumonia. The anæsthesia bronchitis which precedes the pneumonic symptoms, the low temperature range, the absence of bloody sputum, and the course of the attack are significant. In a majority of the cases it is an inhalation pneumonia.

Course and Duration. — Bronchopneumonia is not a self-limited disease. The primary cases, which suggest croupous pneumonia, run a short course and frequently terminate by crisis. The secondary cases

are often prolonged and the defervescence is by lysis. Fatal cases in children often come to an end in from 3 to 5 days. The duration of favorable cases is from 1 to 4 or 5 weeks. Recovery may still take place after an illness of 8 or 10 weeks. In protracted cases tuberculosis is to be feared.

Diagnosis. — DIRECT. — A positive diagnosis cannot be made unless the signs of consolidation are present. Circumscribed areas of relative dulness, usually bilateral with vesiculotympanitic resonance interspersed, together with high-pitched, small mucous and crepitant râles, bronchial breathing, bronchophony, and increased vocal fremitus are diagnostic in the primary as well as in the secondary forms. In cases in which the lesions are massed in a single lobe, careful physical examination will almost always show a focus of consolidation upon the opposite side. The signs of consolidation are often absent, especially early in the attack. The diagnosis is even then probable if in bronchitis the temperature rises to 104° F. (40° C.), the cough becomes short, harassing, and painful, the respiration and pulse-frequency become high, cyanosis develops, and diffuse, high-pitched fine râles are heard.

DIFFERENTIAL.—*Croupous Pneumonia* (see page 124). *Acute Miliary Tuberculosis* (see page 185). *Acute Tuberculous Bronchopneumonia* (see page 200). *Atelectasis.*—The dulness and râles of this condition are modified and sometimes even disappear upon change of posture. Fever is not an essential concomitant. If present it is usually due to an associated bronchitis. *Meningitis.*—In infants marked cerebral symptoms sometimes occur, and the condition closely simulates meningitis. The differential diagnosis between tuberculous meningitis and bronchopneumonia with cerebral symptoms can in some cases only be made by time.

Prognosis.—The outlook is much more favorable in the primary than in the secondary forms. It is greatly influenced by the age of the patient. Within the first year almost every case dies; until the fifth year from 30 to 50 per cent.; in the aged the prognosis is ominous. Pale, fat, flabby children do not bear the disease well. Aspiration pneumonia is a very fatal disease, especially that form which follows operations upon the mouth and throat.

iii. Diseases of the Lungs Characterized by Interstitial Inflammation.

(a) CHRONIC INTERSTITIAL PNEUMONIA.

Cirrhosis of the Lung.

Definition.—Chronic inflammation of the lung with increase of the interstitial tissue and decrease in the vesicular tissue, with general induration and contraction. There are two forms, the local or circumscribed, and the diffuse, involving the greater part of one or both lungs.

1. LOCAL PULMONARY FIBROSIS.—This condition is very common. It occurs as a secondary process in inflammatory diseases of the lung, as bronchitis, croupous pneumonia, and bronchopneumonia. It constitutes an important feature in the complex lesions of tuberculosis and the chief final local process in obsolescent or cured tuberculous disease. It is the

termination of pulmonary atelectasis prolonged through the stages of compression and carnification until the tissue has become organized and expansion is no longer possible. Hence it is a feature of the lung tissue adjacent to aneurisms, tumors, abscesses, hydatids, and gummata. It is especially important as the outcome of neglected pleural effusion, because by the early withdrawal of the exudate the pressure atelectasis may be relieved and subsequent fibroid changes minimized. The clinical phenomena of local fibroid changes are subordinate to those of the primary disease which it accompanies. Retraction of the supra- or infraclavicular spaces, or circumscribed retraction elsewhere, is suggestive. Nevertheless the diagnosis must often be provisional rather than positive. The chief importance of the condition arises from its often constituting the point of departure for fibroid changes involving the entire lung.

2. Diffuse Pulmonary Fibrosis.—To a certain extent the etiological factors are the same as in the localized form. Thus the fibroid changes in chronic bronchitis and emphysema, and associated with bronchiectasis, are usually diffuse. When the condition follows pressure atelectasis it may be diffuse when the greater part of a lobe or lung is compromised. Other conditions which may be followed by chronic interstitial pneumonia are the irritation of a foreign body long retained in a bronchus, chronic bronchopneumonia, in rare instances unresolved croupous pneumonia, pleurisy, the connective-tissue overgrowth invading the lung from a chronic fibrinous pleural exudate—the pleurogenous form of chronic interstitial pneumonia—and syphilis.

Two forms are recognized. (a) *Lobar Interstitial Pneumonia.*—The disease is unilateral. The entire lung is usually involved. It frequently appears as a small, dense, airless mass of tissue lying along the spine. It is densely indurated, showing upon section a compact, grayish, fibroid tissue traversed by the bronchi and blood-vessels. In the pleurogenous form the lung is bound to the wall of the thorax by dense, thick, pleural adhesions. When the process has been intrapulmonary from the beginning the pleural adhesions may be of only moderate thickness. Bronchiectasis is common and often very extensive. In tuberculous cases vomicæ and caseating foci are present and the opposite lung shows evidences of tubercle. The right heart is hypertrophied, and atheroma and local arteriosclerosis are frequently encountered. The heart is displaced toward the affected side. The opposite lung is emphysematous—*vicarious emphysema*—and its border extends beyond the median line toward the affected side. (b) *Disseminated Interstitial Pneumonia.*—The condition is bilateral. There are circumscribed areas of fibrosis separated by lung tissue more or less emphysematous—*collateral vicarious emphysema.* These fibroid islets are deeply situated in the lung tissue and more frequently in the lower lobes. They surround bronchiectatic dilatations.

Symptoms.—Fever when present is due to an acute associated process, or to tuberculosis. There are cough, dyspnœa upon exertion, and expectoration, which may show the special characteristics of the sputum of bronchiectasis. Hæmoptysis is fairly common, especially in the tuberculous cases. The affection is the very type of a chronic disease but the adaptations are remarkable. The general health is often remarkably good when

the extent and nature of the lesions are considered, and the patients are capable of conducting their affairs.

Physical Signs.—Inspection.—The deformity of the chest in well-developed cases is conspicuous both in the unilateral and bilateral types of the affection, though in the latter retraction is more or less modified by compensatory emphysema. In the unilateral variety the affected side of the thorax is retracted and immobile. The ribs approach each other so that the intercostal spaces are obliterated, or the ribs may overlap. The shoulder droops and there is lateral curvature of the spine, the concavity in the dorsal region being toward the affected side. The respiratory muscles and those of the shoulder and arm are wasted. The semicircumference of the affected side is much diminished and remains uninfluenced by respiratory effort. The heart is strongly displaced toward the affected side, being drawn over by the contracting lung. The changes in the cardiac phenomena are much more apparent when the left lung is involved. Under these circumstances there may be an extensive area of pulsation in the second, third, and fourth interspaces to the left of the sternal border. When the right lung is affected the cardiac impulse may be wholly obliterated by the extending median border of the emphysematous left lung. The unaffected side is in strong contrast with its large size, wide intercostal spaces, and free respiratory excursus. Palpation.—If the bronchi are unobstructed the vocal fremitus is increased over the affected side. There is usually epigastric pulsation transmitted from the right heart. Cardiac pulsation is well felt in the second, third, and fourth interspaces in left-sided fibrosis, and in some cases there is a short diastolic thrill to the left of the pulmonary area. Percussion.—Dulness is usually marked and may be extreme at the apex and base. When the bronchi are extensively dilated the percussion sound has the tympanitic quality. This may be especially marked in the axillary region. Over the opposite lung there is hyperresonance. Upon linear percussion the anterior border of the unaffected lung is found to be displaced as far as, or even beyond, the sternal border on the affected side, and its lower border displaced downward, while the lower border of the affected lung and the related organ, *i.e.*, liver or spleen, are displaced upward and show no respiratory movement. Auscultation.—There is more or less widely extended bronchial respiration. At the apex the quality may be cavernous or amphoric; at the base feeble and distant. In some cases small mucous râles are heard. There is bronchophony and, if bronchial dilatation extends to the periphery of the lung, whispering pectoriloquy may be heard. The pulmonary second sound is accentuated and endocardial murmurs frequently appear toward the close of the disease as dilatation and failure of the right heart come on.

Diagnosis.—In the disseminated form the diagnosis may be obscure. The symptoms and signs may be simply those of bronchiectasis. In the unilateral form the diagnosis is unattended with difficulty. The underlying condition cannot, however, always be determined. The resemblance to fibroid phthisis is often very close. Signs of disease in the opposite lung, especially at the apex, fever, hæmoptysis, and foci of moist râles are in favor of a diagnosis of tuberculosis. Tubercle bacilli in the sputum are conclusive.

Prognosis.—The outlook as regards life is favorable; as regards recovery hopeless. The patients live many years and often have no great inconvenience except from cough, expectoration, and dyspnœa upon exertion. The powers of resistance are diminished and they readily succumb to intercurrent disease. Otherwise death is commonly due to progressive failure of the right heart or amyloid disease.

(b) PNEUMONOCONIOSIS.

Definition.—Disseminated fibrosis of the lungs caused by the habitual inhalation of a dust-laden atmosphere in various occupations.

Etiology.—Pneumonoconiosis is the very type of an occupation disease. Several varieties are described, according to the character of the work and the nature of the dust inhaled: *anthracosis* or coal miners' disease; *siderosis*, the form caused by inhaling metallic dust, especially iron oxide, and brass and bronze particles; *chalicosis*, due to mineral dusts, as stonecutters' consumption or the grinders' rot of the workers in cutlery. Similar affections occur in workers in flax and cotton and in grain shovellers.

The condition of the lungs in advanced cases of the disease caused by different substances is practically the same. The interstitial inflammation starts from the peribronchial lymph-nodes in which the dust particles excite proliferation of connective tissue, and in the early stages of the process, especially in anthracosis, is confined to these tissues. There is an associated chronic bronchitis to which many of the symptoms are due. Bronchiectasis is common and in a majority of the cases the clinical picture is that of a chronic bronchitis with emphysema. In anthracosis there is a carbon-laden black spit. In advanced cases softening occurs in the indurated nodules, and small cavities are formed which in some cases suppurate and discharge a purulent fluid by way of the bronchi. Notwithstanding the prevalence of anthracosis among coal miners tuberculous phthisis is comparatively rare.

Symptoms.—The disease does not show itself until after long exposure to the dust. There are the general signs of failing health, with cough, expectoration, often abundant, dyspnœa, and wheezing, especially upon exertion. The mucopurulent sputum in anthracosis is blackish. It is popularly known as "black spit." That of the other forms is light or grayish in color, without gross characteristics. In chalicosis glittering crystalloid particles of silicious material may be seen. Under the microscope the dust particles are seen in the alveolar epithelium. Tuberculosis may form the terminal condition.

Diagnosis.—The direct diagnosis is not as a rule difficult. The anamnesis, the gradual development of the affection after years of exposure, the symptoms of chronic bronchitis with emphysema and bronchiectasis, the mucopurulent sputum with the special characteristics mentioned, and the absence of tubercle bacilli are all of diagnostic importance.

Prognosis.—The outlook is favorable if the condition is recognized early and the patient can change his work. Otherwise the cases run a progressive though very chronic course.

iv. Diseases of the Lungs due to Suppuration and Necrosis.

(a) PULMONARY ABSCESS.

Definition. — Localized collections of pus in cavities formed by the disintegration of lung tissue.

Etiology. — Pulmonary abscess is a secondary process due to the intense action of various pyogenic organisms, among which streptococci and staphylococci are the most common. It may occur under the following conditions:

1. Acute Inflammation.—Suppuration may follow croupous pneumonia. It occurs in two forms: first, the purulent infiltration which constitutes an advanced stage of gray hepatization, and second, the much more rare condition of actual abscess cavity. The latter are usually small, multiple, with shreddy walls, and frequently contain necrotic tissue. They tend by fusion to form larger abscesses. Purulent infiltration and abscess are sometimes present in the same lung. Abscess is more common in bronchopneumonia, especially the form known as aspiration or deglutition pneumonia. In the low fevers and stuporous and comatose conditions, after wounds of the neck and operations upon the nose, throat, and mouth, and suppurative diseases of these parts, an intense acute bronchitis frequently arises, which by extension involves the distant tubules and causes purulent bronchopneumonia. Multiple abscesses, mostly minute but frequently attaining the size of an orange, may result. A similar condition may follow the inflammation caused by a foreign body lodged in a bronchus. Pulmonary abscess is a relatively common sequel of epidemic influenza. 2. Traumatism.—Perforation of the lung from without, as in stab or gunshot wounds, laceration of the lung by a fractured rib, and analogous accidents, may cause pulmonary abscess. 3. Perforation from Within.—Sudden invasion of the lung by purulent or otherwise infected substances from adjacent organs is a common cause of abscess. Cancer of the œsophagus, abscess of the liver, a suppurating hydatid cyst, or the aspiration of the pus in empyema suddenly rupturing into the lung may cause abscess. 4. Infective Emboli.—Metastatic abscesses in the lung are common in septic—pyæmic—states. The purulent foci are multiple, mostly subpleural, and at first wedge-shaped. They are commonly small, but occasionally extensive purulent infection occurs. The related pleura is inflamed and covered with a thick, greenish lymph. Occasionally softening and perforation are followed by pneumothorax. 5. Tuberculous Abscesses.—Circumscribed local suppurative processes enter largely into the complex lesions of pulmonary tuberculosis, especially in the later course of the disease. They are associated with caseation and cavity formation, and give rise to important and significant symptoms and physical signs.

Symptoms.—As in other suppurative processes there are irregular chills and fever. In pneumonia the general symptoms are aggravated, the sputum becomes purulent, and the signs of a cavity can sometimes be demonstrated. In pyæmia the local symptoms of pulmonary abscess are obscured by the general symptoms of sepsis, and the condition is usually

overlooked. Aside from the presence of pus in large amounts the sputum presents characters of diagnostic importance (see Part III, page 473).

Physical Signs.—The signs of a cavity which empties itself under the stress of violent paroxysms of cough, and again refills, may be elicited upon physical examination when the abscess is of sufficient size and situated in the periphery of the lung. In small abscesses the signs are often wholly inconclusive.

Diagnosis.—DIRECT.—When the above symptoms and signs are present, especially when the etiological factors essential to diagnosis can be established and purulent sputum containing elastic fibres and the other characters named accompanies the cough, the diagnosis is positive. In many cases, however, it is at best provisional, and not rarely abscess in the lungs not suspected during life is found upon the post-mortem table.

DIFFERENTIAL.—*Collections of Pus Perforating into the Lung.*—Accumulations that may break into the lung occur in empyema, subphrenic abscess, hepatic abscess, suppurating hydatid cyst, in spinal caries, and other suppurative processes in adjacent viscera. The differential diagnosis rests upon the presence of symptoms and signs significant of such pathological conditions, and especially upon the absence of the elastic fibres from the expectorated pus. *Bronchiectasis.*—The etiological factors are different. Both are secondary affections, but bronchiectasis is a chronic, pulmonary abscess an acute, affection. The cough is more urgent and less constant in bronchiectasis, and the sputum more foul and does not as a rule contain elastic fibres, which, when present, are less abundant than in abscess. *Pulmonary Cavities in Tuberculosis.*—Etiological considerations are important. The slow development of phthisical cavities, their antecedent and concomitant phenomena, the characters of the sputum, and the presence of tubercle bacilli are of positive diagnostic significance. *Pulmonary Gangrene.*—The sputum in gangrene of the lung has an extremely intense, putrid odor not often present in the purulent expectoration of pulmonary abscess, and contains shreds of decomposing lung tissue, which can be readily detected when it is spread out upon a glass plate. These shreds frequently present the structure of the pulmonary alveoli.

(b) GANGRENE OF THE LUNG.

Definition.—Decomposition of lung tissue caused by the action of the bacteria of putrefaction. It may be diffuse or circumscribed.

Etiology.—Sphacelus of the lung occurs as an anatomical condition under a variety of circumstances. Impaired vitality of the tissues from general or local causes is a necessary predisposing factor. The subjects are commonly greatly debilitated by long-continued chronic or grave acute disease. Severe general disturbances of nutrition, such as are caused by prolonged infections or disease of the bones, or arise in malignant disease or diabetes mellitus, constitute predisposing influences of importance. Equally important are bronchiectasis, especially that form of bronchiectasis which occurs as the result of the pressure of a tumor or aneurism, tuberculous cavities and putrid bronchitis, since the putrid contents of the cavities or bronchi often cause secondary gangrene when aspirated

into the adjacent tissues. Among the acute diseases in which gangrene of the lung occurs are croupous pneumonia, particularly when it affects debilitated or diabetic persons; aspiration bronchopneumonia, whether the infectious material be derived from the nose or throat of the patient, or from some focus of disease adjacent to the lung, as cancer of the œsophagus, abscess of the liver, or empyema; embolism of the pulmonary artery, especially when the embolus is infected, as in pyæmia or necrosis of the bones, and in enteric fever thrombosis of one of the principal branches of the pulmonary artery. No satisfactory explanation of the occasional occurrence of gangrene under these circumstances, when it remains absent in the majority of similar cases, has been advanced.

1. Diffuse Pulmonary Gangrene.—This form is very rare. It has been observed after croupous pneumonia and in the most intense cases of aspiration pneumonia. It may also occur after the occlusion of a large branch of the pulmonary artery by an embolus or thrombus. A large portion or the whole of a lobe may undergo putrefaction in the course of a day or two.

2. The Circumscribed Form.—The gangrenous portions of lung are surrounded by an intensely congested and œdematous border of tissue. The lesions may be single or multiple. They are more commonly situated in the lower lobes than the upper and in the peripheral than in the central parts of the lung. The greenish-black gangrenous tissue rapidly softens, with the formation of an irregular cavity with shreddy walls and an abundant, horribly offensive fluid.

Symptoms.—The expectoration is profuse, thin, and green or brownish in color. Its odor is exceedingly fetid, disgusting, and penetrating. In some instances it is mawkishly sweet. It is not only present upon the breath but also pervades the atmosphere of the patient's room (see Part III, p. 457). The symptoms of the primary affection and constitutional debility usually precede the characteristic phenomena of gangrene of the lung. There is commonly a moderate fever associated with rapid pulse and great general depression. Hemorrhage is not uncommon and may prove fatal.

Physical Signs.—The signs in the diffuse form are those of infiltration, impaired resonance, with the tympanitic quality, and vesiculobronchial or bronchial respiration. In the circumscribed form the signs of a cavity predominate. Very often in limited gangrene the signs may elude detection. Severe bronchitis is common.

Diagnosis.—Direct.—The sputum and the odor of the breath are characteristic. The presence of shreds of gangrenous alveolar tissue is conclusive.

Differential.—From putrid bronchitis and bronchiectasis gangrene of the lung cannot always be differentiated, except by the presence of lung tissue in the sputum, and when this is found in old standing cases it constitutes positive evidence of secondary gangrene.

Prognosis.—Pulmonary gangrene is almost always a terminal event. The patient sinks rapidly and dies from exhaustion. When the gangrenous area can be localized, and the condition of the patient is favorable, a surgical operation may be the means of saving life. In rare cases of circumscribed gangrene encapsulation has taken place, and still more rarely recovery has occurred after the expectoration of pieces of gangrenous lung of considerable size.

v. New Growths in the Lungs.

Neoplasms in the lungs are usually malignant. They are primary or secondary. The former are rare; the latter comparatively common.

1. Primary Tumors.—Carcinoma is the usual form. Much more rare are primary sarcoma and endothelioma. The growth usually invades one lung and forms a large mass, which may ultimately break down and give rise to a cavity. Diffuse cancerous infiltration may simulate tuberculous bronchopneumonia. Diffuse miliary infiltration has been described —*carcinosis pulmonum miliaris.*

2. Secondary New Growths.—Every variety of malignant growth may by metastasis invade the lungs. In comparatively rare instances the secondary growth may be solitary, and chiefly involves the pleura. Usually they are multiple and occupy both lungs. Large tracts of pulmonary tissue may be densely invaded. The primary tumor is usually mammary, in the gastro-intestinal or genito-urinary tract, or in the bone. It may be epithelioma, scirrhus, colloid, sarcoma, enchondroma, or osteoma. In melanosarcoma the primary growth may have its starting-point in a pigmented mole. In Hodgkin's disease the growth may perforate the sternum and widely involve the lungs, or it may reach the lungs by way of the tracheal and bronchial lymph-nodes. In cancerous disease there is commonly secondary implication of the tracheal and bronchial glands, and less often of the cervical chains. Other superficial glands, including the inguinal, may be enlarged. Pleurisy is common. It may be cancerous or serofibrinous. Frequently both conditions are present. Not rarely the effusion is hemorrhagic.

Etiology.—New growths in the lungs occur with greatest frequency in middle life. Males suffer more commonly from primary malignant disease of the lungs and pleura; women from the secondary form.

Symptoms.—The clinical picture is not well defined, especially in the primary form. It sometimes suggests chronic pneumonia, sometimes a mediastinal tumor or thoracic aneurism, or again there may be nothing to suggest an affection of the lungs. Pain is usually present when the pleura is affected. It may be substernal. Cough is not constant. It is frequently aggravated in certain postures and may be dry and attended with pain. There is in many cases a jelly-like bloody sputum which is highly suggestive, though it may occur in other conditions. Dyspnœa upon exertion is a symptom of large growths or extensive infiltration, and may be paroxysmal. Pleural effusion, often found upon aspiration to be hemorrhagic, may develop. The lymph-nodes of the axilla or above the inner end of the clavicle are frequently enlarged. There is progressive wasting and cachexia, and toward the end irregular fever of remittent type. Pressure symptoms are common. These consist of distention of the large veins, with cyanosis of the face and one or both arms, enlarged and tortuous veins over the upper part of the chest, distressing dyspnœa or stridor from compression of the trachea or large bronchi, brassy cough, and aphonia from pressure upon recurrent laryngeal nerves. In large unilateral tumors the heart is displaced toward the opposite side and the diaphragm depressed.

Physical Signs.—The signs may be due to the tumor itself or to an accompanying pleural effusion. In the latter case the affected side is enlarged, the intercostal depressions obliterated, the respiratory movement restricted. The vocal fremitus is diminished. Upon percussion and auscultation the signs are modified by the presence of fluid and its amount, which is not usually great. Even when it is considerable, the tumor affects its distribution and upper lines. There is dulness which may be complete at the base. The breath sounds are feeble and distant. There may be well-characterized bronchial breathing.

Diagnosis.—Direct.—In primary cases the diagnosis may be difficult or impossible. Important criteria are strictly unilateral phenomena, irregular character and distribution of the physical signs, dark, jelly-like, mucoid, bloody expectoration, tendency to cachexia, and implication of superficial lymph-nodes. If carcinomatous tissue elements are found in the sputum or the growth perforates the chest wall—very rare events—the diagnosis is positive. The X-ray examination is important. In a case of single large growth in the lower lobe of the right lung, with multiple pigmented cutaneous lesions, occurring in a man aged sixty-four, the results of physical examination were fully confirmed. In the secondary form a probable diagnosis may be made with some confidence when the above described pulmonary symptoms arise in the course of several months after the recognition of a primary malignant tumor, as of the breast, womb, stomach, or bowel, with or without operation.

Differential.—The recognition of a mediastinal new growth or an aneurism of the aorta, in contradistinction from new growths in the lungs, may involve serious diagnostic difficulties.

Prognosis.—The course of malignant growths in the lungs is lethal, the end occurring within a year or two, and not rarely within a few months after the appearance of the symptoms.

V. DISEASES OF THE MEDIASTINUM.

Enlarged lymphatic glands, suppurative lymphadenitis, abscess, interstitial emphysema, chronic indurative mediastinitis, and new growths are to be considered.

1. **Enlargement of the lymphatic glands** in the mediastinum accompanies inflammation of the bronchi, bronchopneumonia and the specific infections, croupous pneumonia, measles, pertussis, and tuberculosis. This constant anatomical change does not commonly attain sufficient dimensions to cause definite symptoms or physical signs; exceptionally pressure upon the trachea may cause a paroxysmal, brassy cough, and dulness over the manubrium sterni, in the upper part of the interscapular region, or a modification of the sound upon direct percussion over the upper dorsal spines.

2. **Suppurative Lymphadenitis.**—Suppuration may occur in the tracheal or bronchial lymphatic glands as the result of ordinary inflammation. More commonly it is of tuberculous origin. The symptoms are obscure. Perforation into a bronchus, the œsophagus, or the aorta may occur. In other cases the fluid contents of the glandular abscess are absorbed, lime salts are deposited, and the condition becomes one of anatomical rather than clinical interest.

3. **Mediastinal Abscess.**—The abscess cavity usually occupies the anterior mediastinum. The condition may be acute or chronic. The acute cases are due to traumatism or occur in connection with the acute febrile infections; the chronic cases are as a rule tuberculous. Males are more commonly affected. The pus shows a disposition to burrow and may find its way through an intercostal space, into the abdomen, may rupture into the trachea or œsophagus, or may perforate the sternum.

Symptoms.—Substernal pain is constant. In the acute cases it is severe and throbbing, and accompanied with chills, fever, and sweating. Large abscesses may give rise to pressure symptoms and dyspnœa. There may be resorption of the fluid and inspissation.

Physical Signs.—A fluctuating tumor may appear in the episternal space or at the sternal border. In the latter situation it may suggest empyema necessitatis. Pulsating synchronously with the heart it may simulate a pulsating empyema or an aneurism. From pulsating empyema it may be distinguished by the absence of the signs of pleural effusion; from aneurism by absence of murmur, of diastolic shock, and of the expansile character in the pulsation. Exploratory puncture with a fine needle may be performed.

4. **Emphysema.**—The escape of air into the connective-tissue spaces of the mediastinum is an occasional event after traumatism or surgical operations upon the neck, as tracheotomy, and in pertussis. It may be associated with pneumothorax. There are no special symptoms. Crackling râles having the rhythm of the heart are heard over the sternal region. If the air finds its way into the subcutaneous tissue of the neck the crepitus may be recognized upon kneading the tissues lightly with the finger-tips. Etiological factors are important.

5. **Indurative Mediastinitis.**—Adherent pericardium with chronic proliferative mediastinitis is a rare condition. The heart is greatly enlarged. Its action is hampered by the extensive adhesions to the adjacent parts; its signs obscured by the greatly thickened fibrous tissue of the mediastinum. Friction sounds may sometimes be heard along the sternal borders. The nutrition of the heart muscle ultimately fails, and dyspnœa, cyanosis, and anasarca develop.

6. **Mediastinal New Growths.**—The common varieties are carcinoma and sarcoma. Dermoid cysts, hydatid cysts, and lymphomata, fibromata, lipomata, enchondromata, and gummata are of comparatively infrequent occurrence. The tumor may have its origin in the thymus, the lymph-glands, or the pleura or lung. Tumors of the anterior mediastinum originate from the remnants of the thymus of the connective tissue; those of the middle and posterior mediastinum from the lymph-nodes. Primary tumors are more commonly sarcoma than carcinoma. Among personal predisposing influences age and sex are important. Mediastinal tumor most frequently develops in the fifth decade of life and in men.

Symptoms.—The important manifestations of a tumor developing in or encroaching upon the mediastinal spaces are displacement and pressure symptoms. These symptoms relate to the heart, great vessels and nerves, the trachea, bronchi, lungs, and pleuræ, and the œsophagus. The symptoms depend upon the size of the new growth, and its immediate

location. A small tumor may not be the occasion of any derangement of function. On the other hand, as the growth increases, remarkable adaptations take place, and life may be maintained despite great compression and dislocation of the heart and lung. The pressure of the tumor exerted upon the wall of the heart interferes with diastole and diminishes the volume of blood thrown into the aorta with the ventricular systole. The pulse is therefore small and frequent. The dislocation of the organ is backward or downward in tumors of the anterior mediastinum, with displacement of the apex beat to the left. The liver or spleen is also displaced downward by large mediastinal tumors. There may be cardiac dyspnœa from pressure. The presence of the tumor interferes with respiration and produces of itself a slight degree of cyanosis, which is increased by pressure upon large venous trunks. When the intrathoracic portion of the inferior vena cava is affected, venous distention and œdema of the abdomen and lower extremities result; when the superior vena cava is compromised, swelling and œdema in the face and both upper extremities may occur, together with signs of interference with the cerebral circulation, such as headache, vertigo, and ringing in the ears. A collateral venous circulation may be established in either case with great distention of the superficial veins of the abdomen or thorax. Pressure upon and obstruction of the right or left innominate vein is more common. This hinderance to the return of the venous blood manifests itself by œdema of the face and arm of the corresponding side, and enlargement of the superficial veins of the thorax. The arterial trunks yield less readily to the presence of the tumor than the veins. When the pressure becomes so great as to interfere with the lumen of the subclavian or innominate, the pulse upon the affected side is enfeebled. Pressure upon the recurrent laryngeal nerves causes dyspnœa, aphonia, and the severe, brassy, paroxysmal cough often present. The laryngoscope should be used. Pressure upon the phrenic may cause hiccough and shallow respiration; upon the vagus asthmatic attacks and dysphagia in the absence of direct pressure upon the œsophagus, and bradycardia or tachycardia. Implication of the sympathetic may cause dilatation or contraction of the pupil upon the affected side—inequality of the pupils. Dyspnœa is an early and constant symptom. It is due to various factors, as pressure upon the heart, the recurrent laryngeal nerves, the trachea, the lungs themselves, or the presence of a pleural effusion. It is often slight when the patient is quiet, but severe and distressing upon exertion. The mechanical compression of considerable portions of a lung manifests itself in dyspnœa of inspiratory type with slow and deep respiratory movements. In extreme conditions of pressure there is orthopnœa. When the pressure is unilateral there may be inspiratory depression of the intercostal spaces. Areas of fibrinous pleurisy are attended with local pains and friction sounds. Compression of the œsophagus renders the act of swallowing difficult or in extreme cases impossible.

Physical Signs. — *Inspection.* — Orthopnœa, œdema, and cyanosis of the face, arm, and upper part of the chest, and varicose enlargement of the superficial mammary and epigastric veins are often seen. But in many cases these signs are absent. In old cases there may be clubbing of the finger-tips and incurvation of the nails. There may be bulging of the

sternum. The new growth may even erode the bone or perforate the chest wall at the sternal border. The impulse of the heart may be displaced to the left and downward. Respiratory derangements are conspicuous. In some cases inspiratory retraction of the soft parts of one side denotes unilateral compression of a main bronchus or the large part of the lung. Diminished respiratory excursus upon one side may be a sign of pleural effusion. *Palpation.*—Vocal fremitus is absent over the tumor and over pleural effusions. If the tumor pulsates it lacks the forcible, expansile impulse of an aneurism. *Percussion.*—Dulness of a high grade and increased resistance are present over the tumor. The borders of the area of dulness are irregular, and do not correspond to the outline of the heart or of the margin of an infiltrated lung. The dull area is continuous with the dulness of a pleural effusion when the latter is present and its persistence after the withdrawal of the fluid is of diagnostic significance. *Auscultation.*—The respiratory murmur retains its vesicular quality, in the main, but is enfeebled. At the borders of the tumor it may, owing to pressure atelectasis, have the bronchial quality. Stridor is the sign of compression of the trachea or a main bronchus.

Diagnosis.—DIRECT.—An area of dulness in the sternal region, with irregular and advancing borders, bulging of the breast-bone, dyspnœa of inspiratory type, absence of respiratory murmur, displacement of the heart and of the liver or spleen, signs of obstruction of the venous circulation, of pressure upon the vagi, the sympathetic, the phrenic, and the recurrent laryngeal nerves, the trachea, bronchi, or the lung itself, and dysphagia constitute a symptom-complex upon which the diagnosis of mediastinal tumor may be confidently made. The association of a number of them justifies a provisional diagnosis. When, however, to several of these signs are added the presence of enlarged superficial lymph-nodes and the visible and palpable evidences of a tumor perforating the sternum or the chest wall at the borders of the sternum, or advancing into the episternal notch, the diagnosis becomes positive.

DIFFERENTIAL.—Obscure pressure symptoms may arise in consequence of the presence of a small mediastinal tumor, tuberculous tracheal or bronchial glands, gummata or syphilitic cicatrices, or a small aneurism. The differential diagnosis of these conditions cannot be made nor can a positive diagnosis of mediastinal tumor be reached until abnormal dulness associated with distinct evidences of intrathoracic pressure appear. With reference to the differential diagnosis, pericardial and pleural effusions, malignant disease of the pleura, and aneurism of the aorta demand special consideration. *Pericardial Effusion.*—Fluid pericardial and pleural exudates progressively displace the organs contained in the mediastinum; mediastinal new growths compress them. A flat percussion sound is the sign of both conditions; but the area of flatness in uncomplicated effusion into the serous sacs has definite and regular outlines, whereas in mediastinal tumor its borders are irregular and anomalous. In pericardial effusion the apex beat may be faintly palpable within the dull area and more distinct when the patient bends forward. In this attitude the dulness is also slightly increased in its transverse diameter. In mediastinal tumor, on the other hand, the cardiac impulse is at the left border of the dull area, which is

not affected by change of posture. In cases in which the tumor extends between the apex of the heart and the wall of the chest no impulse can be detected. *Pleural Effusion.*—This condition is frequently associated with mediastinal tumor. Under these circumstances the physical signs are anomalous. The persistence of dulness of irregular outline and compression symptoms after the withdrawal of the fluid are significant. Dyspnœa upon exertion, and dislocation of adjacent organs, unilateral prominence of the chest wall with diminished respiratory excursus upon the same side, point to pleural effusion. In tumor the vocal fremitus is more commonly preserved, signs of pressure upon the recurrent are more frequent and more marked, and dysphagia is often present. An exploratory puncture will at once clear up any uncertainty. *Malignant Disease of the Pleura.*—More obscure is the differential diagnosis when this condition is present, especially if the new growth takes origin from the costal pleura and has attained considerable size. These tumors cause local bulging of the chest wall, dulness of irregular outline, effusion into the pleural sac, and compression of the lung, large venous trunks, and the œsophagus, and they may invade the mediastinal spaces. In the latter case they constitute a variety of mediastinal tumors. *Aneurism of the Thoracic Aorta.*—The differential diagnosis between mediastinal tumor and aneurism may be very difficult, especially in areas in which the outline of the aneurism is irregular and the sac more or less occupied by firm clot. The symptoms of both are due to pressure, and the effects of pressure are practically the same, whether it be exerted by a solid tumor or one distended with blood. The following data are in favor of mediastinal tumor: marked cyanosis, venous engorgement, and the enlarged superficial veins of collateral circulation; right-sided œdema of the face and arm; absence of diastolic shock; absence of tracheal tugging; relatively short duration; if pulsation be present, it is limited in extent, especially in large tumors, and not expansile in character; if murmurs be present, they are usually systolic only; enlargement of the lymph-nodes and a firm nodular, rather than an elastic, mass palpable at the sternal notch. The following render the diagnosis of aneurism probable: absence of œdema and cyanosis, and the absence of enlarged superficial veins upon the thorax and abdomen; the presence of diastolic shock and tracheal tugging; prolonged duration; expansile, heaving, and forcible pulsation, either in areas in which the tumor lies in relation with the chest wall or has perforated the sternum; double murmurs, inequality of the radial pulses, and severe boring pain radiating to the back, arms, and neck. Loud murmurs heard over a considerable area are common in aneurism, but murmurs may be wholly absent, whereas in mediastinal tumor murmurs are only occasionally heard.

Examination by the X-rays yields conclusive results when the shadow shows in any part the rounded expansile pulsation of an aneurismal sac. Rest in bed, a limited diet, the restriction of fluid, and the administration of potassium iodide may be followed by relief of pain in aneurism.

Diagnosis of the Location of Mediastinal Tumors.—Developing in the anterior mediastinum, new growths push forward and sometimes erode the sternum. They may frequently be felt in the suprasternal notch. The symptoms indicate compression of venous trunks. Dyspnœa is often

urgent, and the lymphatic glands of the neck are often enlarged. When the growth occupies the middle and posterior mediastinum the physical signs may be obscure. Pressure is especially exerted upon the œsophagus and the recurrent laryngeal nerves. Dysphagia, urgent dyspnœa, and a brassy, laryngeal cough occur. In tumors springing from the pleura or lung symptoms of pressure upon the blood-vessels, nerves, and gullet are less marked; signs indicating compression of the lung itself more prominent. A complicating pleural effusion is common. There is a tendency to rapid emaciation and cachexia.

Diagnosis of the Character of Mediastinal Tumors.—The most common forms are carcinoma and sarcoma, the former developing as a rule later in life than the latter, but there are many exceptions to this statement. Rapidity of growth, metastatic tumors, glandular enlargement, loss of weight, anæmia, cachexia, subnormal temperature, and cutaneous pigmentation point to carcinoma. In sarcoma the weight and the appearance of fair nutrition may be long maintained.

Prognosis.—Acute enlargement of the tracheal and bronchial lymphatic glands usually subsides with the pulmonary disease of which it is a feature. Suppurating glands may rupture into contiguous structures or undergo retrogressive changes, with resorption of the fluid and deposition of lime salts. Tuberculous glands remain enlarged, with the tendency to caseation and softening. Abscess is a serious affection, but a fair proportion of the cases are amenable to surgical treatment. Chronic proliferative mediastinitis is a progressive disease tending to destroy life by impairing the function of the heart. The conditions under which mediastinal emphysema occur are usually of grave prognostic import. Finally, mediastinal tumors are in a large proportion of the cases malignant and without hope.

VI. DISEASES OF THE PLEURA.

i. Pleurisy.

Definition.—Inflammation of the pleura. The cases may be grouped according to various principles of classification. Etiologically primary and secondary forms may be recognized, but this distinction cannot always be made at the bedside; clinically the disease may run an acute or chronic course, but forms characterized by similar features differ greatly in intensity and duration. The anatomical division into dry or plastic pleurisy and pleurisy with effusion is most convenient for descriptive purposes.

(a) Fibrinous or Plastic Pleurisy.

Pleuritis Sicca.

Acute Dry Pleurisy.—The pleural membrane is the seat of a fibrinous exudate of varying thickness, arranged in a single layer or in superimposed strata. This form of pleurisy apparently occurs in some instances as a primary disease after exposure to cold or contusion of the chest. It is far more frequent as a secondary affection in acute and chronic diseases of the lung when the lesions extend to the pleura. It is an almost constant

accompaniment of croupous pneumonia and very common in bronchopneumonia. Pulmonary infarct, abscess, gangrene, and malignant disease cause inflammation of the pleura when they extend to the periphery of the lung. Dry pleurisy is a constant accompaniment of chronic pulmonary tuberculosis, alike when the primary infection involves the lung or the pleura itself.

Symptoms.—The subjective symptoms of pleural irritation are present, namely, the pain known as *stitch in the side*, and dry cough. Fever of moderate intensity is usually also present. The pain is usually referred to the region of the nipple or the axilla. In diaphragmatic pleurisy the pain is often referred to the abdomen, especially in children. It is increased upon deep breathing. The respiratory movement is therefore consciously or unconsciously somewhat restricted, and the patient presses his hand upon the affected side when he coughs. Cough is sometimes absent.

Physical Signs.—*Inspection.*—The respiratory movement is somewhat limited upon the affected side. *Palpation.*—A distinct friction fremitus may very often be felt. This sign is due to roughening of the opposed pleural surfaces. *Percussion.*—There is no change in the sound, but upon linear percussion a limited inspiratory descent of the lower border of the lung upon the affected side may be made out. *Auscultation.*—A pleural friction rub, the almost constant and always distinct sign of dry pleurisy, is heard. When the plastic exudate affects the pleura in the neighborhood of the heart the friction sounds occur, not only synchronously with the respiratory movements, but also with the cardiac revolution—*pleuropericardial friction.* In miliary tuberculosis involving the pleura a fine, widely diffused friction sound may be heard.

Diagnosis.—DIRECT.—The friction sound is of positive diagnostic significance. When it is not heard the diagnosis of fibrinous pleurisy cannot be affirmed. Pain in the side, increased upon cough and deep breathing, occurs in other conditions. Even when the friction sound is present, we cannot in every case be sure that the pleurisy of which it is the sign is not associated with pneumonia, or the forerunner of pleural effusion. Time is therefore in certain cases essential to the diagnosis.

DIFFERENTIAL.—*Pleurodynia.*—Myalgia of the intercostal muscles of one side, intensified by cough and deep breathing, and by pressure often over a circumscribed area, may be mistaken for dry pleurisy. It is more common upon the left side. The absence of friction sounds and the constitutional condition under which myalgias arise render the differential diagnosis an easy matter. *Intercostal Neuralgia.*—The pain is limited to the course of nerve-trunks and is paroxysmal. There are tender points. The disease is common in neurotic and hysterical women. It is a chronic affection. Friction sounds do not occur. *Neuritis of Intercostal Nerves.*—Whether associated with herpes zoster, spinal caries, or disease of the cord, or due to the pressure of a tumor or aneurism, this painful affection of the chest is sometimes mistaken for pleurisy. The one essential diagnostic point is the presence or absence of friction sounds. Causal factors are important, and the distribution of the pain along nerve-trunks with *points douloureux* is significant.

Prognosis.—The outlook is mostly favorable. In the primary form, after a few days adhesions take place, the friction murmur disappears, and the pain ceases. The secondary forms often run the same course.

Chronic Dry Pleurisy.—There are three forms: primitive dry pleurisy, that form which follows the resorption or withdrawal of pleural effusions, and tuberculous dry pleurisy.

1. Primitive Dry Pleurisy.—This variety may develop insidiously without marked symptoms, and be first recognized by the accidental discovery of the friction fremitus, or it may, as is commonly the case, be the outcome of the acute disease. Limited or general pleural adhesions take place. The respiratory function is but little affected. With general bilateral adhesions the respiratory play of the chest is restricted. Percussion is normal. The excursus of the lower border of resonance is diminished and the lessened movements of the diaphragm are confirmed by Litten's sign. This form of pleurisy may result in remarkable thickening and connective-tissue proliferation within the lung, with contraction and induration—*pulmonary cirrhosis*.

2. Adhesive Pleurisy Following the Removal of Exudates.—Upon resorption of an effusion, or its removal by aspiration or otherwise, the pleural surfaces unite and the fibrinous material becomes organized. This process is most marked at the base of the chest and gives rise to a characteristic deformity, in which there is flattening, with narrowing of the intercostal spaces and overlapping of the ribs, deficient expansion, enfeebled respiratory murmur, and dulness. The pathological change is largely due to prolonged pressure atelectasis, a fact of great importance as bearing upon the prompt removal of pleural effusions. The condition follows serofibrinous pleurisy, empyema, and traumatism of the chest, especially gunshot and stab wounds. It is of every grade, from the slight retraction of the chest wall following a rapidly removed serofibrinous effusion, relieved by respiratory gymnastics, particularly in young persons, to the gross and disfiguring deformity which is seen after neglected effusions of all kinds, especially old empyemata, and is associated with a permanently compressed, airless, fibroid and bronchiectatic lung.

3. Tuberculous Dry Pleurisy.—The course of tuberculous dry pleurisy is from the onset essentially chronic and characterized by great thickening, together with implication of the connective-tissue framework of the lung. It may involve both sides and usually begins at an apex. Proliferating pericarditis or peritonitis may be also present.

Flushing or sweating of one cheek and dilatation of a pupil may occur when the pleural thickening implicates the upper thoracic ganglion.

The differential diagnosis between a circumscribed pleural effusion and great pleural thickening is in many cases extremely difficult, a fact not surprising in view of the physical condition and the occasional presence of small collections of residual fluid in the thickened pleura. Dulness, even flatness, feeble respiratory sounds, diminished vocal resonance, and absent vocal fremitus occur in both conditions. The use of the aspirator will usually at once determine the diagnosis.

(b) Pleurisy with Effusion.

Pleuritis Exudativa.

Definition.—Inflammation of the pleura in which fluid exudate is associated with the fibrin.

Pleural inflammation is due to microbic infection. The organisms present in the exudate are, with greatest frequency, the tubercle bacillus, the pneumococcus, and the streptococcus; far less commonly the staphylococcus, Bacterium coli commune, Friedländer's bacillus, the bacillus of Eberth, and the Klebs-Löffler bacillus. Mixed infections occur. According to the character of the effusion the following forms are recognized: serofibrinous, purulent, hemorrhagic, and chyliform.

SEROFIBRINOUS PLEURISY.

This term is not used to designate those cases in which small quantities of serum are entangled in the meshes of a loose plastic exudate, but to describe considerable collections of fluid which, unless prevented by adhesions, accumulate under the influence of gravity in the dependent parts of the pleural sac.

Etiology.—Predisposing Influences.—The cases may be divided into idiopathic and secondary. Idiopathic or primary pleurisy often quickly follows a wetting or chill in an apparently healthy person. The majority of the cases are, however, tuberculous. Serofibrinous pleurisy may follow injury to the chest. With reference to personal predisposition, hospital statistics show a greater liability on the part of males—5 to 1—and in middle life, especially between 40 and 50. The disease occurs, however, at all ages. Secondary pleurisy occurs not only in connection with tuberculous disease of the lung, or tuberculous lesions in distant parts of the body, but also in croupous pneumonia and bronchopneumonia, malignant disease of the pleura or lung, pericarditis, rheumatic fever, enteric fever, diseases of the liver, and chronic nephritis.

Exciting Causes. — Exposure to cold and damp and traumatism lower local tissue resistance to pathogenic micro-organisms.

Morbid Anatomy. — The serous and fibrinous exudates are present in varying proportions. Fibrin may be scanty, or form thick, shaggy layers upon the pulmonary and costal pleuræ and curd-like masses or flocculi which float in the serum and collect in the most dependent part of the pleural sac. The fluid is clear or slightly turbid, according to the relative abundance of cells and fibrin masses which it contains. It is of a pale citron or lemon color, but may be darker. It coagulates on boiling, but sometimes on standing undergoes spontaneous coagulation. Chemically it resembles blood-serum. It may show the presence of cholesterin, uric acid, or sugar. Microscopically there are seen leucocytes, endothelial cells, fibrin shreds, and erythrocytes. The fluid, according to its volume, exerts pressure upon the lungs and adjacent organs. In small effusions the lower lobe is compressed and partially atelectic; in large effusions the entire lung may be reduced to a flat, airless, carnified mass lying against

the spine. In large effusions the mediastinum and heart are displaced toward the opposite side, the diaphragm is depressed, and with it the liver or spleen, as the case may be.

Symptoms.—Serofibrinous pleurisy may begin insidiously or with acute symptoms. The former mode of onset is more common in children and aged persons and in the secondary forms which develop in acute or chronic disease. The chief symptoms are shortness of breath on exertion and rapid anæmia. The latter may be preceded by prodromes, or a chill with fever and pleural pain may suddenly occur. If relatively mild these symptoms suggest acute plastic pleurisy; if severe, croupous pneumonia. The pain is severe, lancinating, and aggravated by deep breathing. It is referred to the nipple or axillary region; sometimes, probably when the diaphragmatic pleura is involved, to the umbilical region or the hypochondriac region of the affected side, suggesting gastralgia, gastric ulcer, or an acute inflammatory infradiaphragmatic inflammation, as cholecystitis or appendicitis. In rare cases the pain is located in the lumbar region. The temperature rises gradually rather than rapidly and attains an average of 102°–103° F. (39°–39.5° C.). The fever is atypical and irregular and of varying duration. Surface observations show in the early course of the disease a slightly higher elevation upon the affected side. Cough as a rule is present. It is accompanied by scanty, mucous expectoration. When this contains blood, a larval pneumonia is to be suspected. Dyspnœa is at first due to the fever and pain; later to circumscription of the respiratory surface in consequence of the compression of the lung. The more rapidly the fluid accumulates the more urgent the shortness of breath. A large effusion if slowly formed may cause little or no dyspnœa so long as the patient lies quietly in bed. A moderate leucocytosis—12,000 to 15,000—is present during the febrile period. In a small proportion of the cases the leucocytes are below normal.

Physical Signs.—Inspection.—The patient prefers to lie upon his back slightly propped up on pillows or upon the affected side. In large effusions the contour of the affected side appears to be abnormally full, and the chest may show upon measurement, due allowance being made for the normal disparity, an increase of 2 or 3 cm. in the semicircumference. The intercostal furrows are absent. The immobility of the affected side is often in striking contrast to the movement of the sound side, which is exaggerated by vicarious function. In right-sided effusion the apex of the heart may be displaced to the fourth interspace beyond the mammillary line, or even as far as the left anterior axillary line; in left effusions the apex may lie behind the sternum and no impulse be seen; or there may be a visible impulse in the third or fourth interspaces as far to the right as the nipple line. Palpation.—The signs obtained upon inspection are confirmed by the sense of touch. The vocal fremitus is diminished or absent. In children the fremitus attendant upon crying is sometimes transmitted along the chest wall to the affected side. It may be present in circumscribed areas over large effusions when there are localized old pleural adhesions. Fluctuation is not a sign of simple serofibrinous pleurisy, and œdema of the chest wall scarcely ever occurs. Mensuration.—The difference in the contour between the sides as determined by

the cyrtometer is very striking. There is an increase in the anteroposterior diameter, together with an increase in the semicircumference. The difference in respiratory expansion may be accurately measured by the saddle-tape. Percussion.—The percussion sound over the effusion is flat, and the percussing finger perceives an absence of elasticity which is very suggestive. Above the level of the fluid, Skodaic resonance—the sign of relaxation of vital intrapulmonary tension—may be elicited in front and to a less degree behind. The upper line of flatness is not horizontal, but rises in a curve resembling the italic letter *S*, starting at its lowest point from the spine and rising to the axilla, from which it descends obliquely in a straight line to the sternum. This line, known as "*Ellis's line of flatness*," has been established by abundant clinical and experimental studies. It is much modified when the patient has been confined to bed during the accumulation of the fluid, when there are lesions in the lung which modify its shape and consistence, and when the compression of the lung is inter-

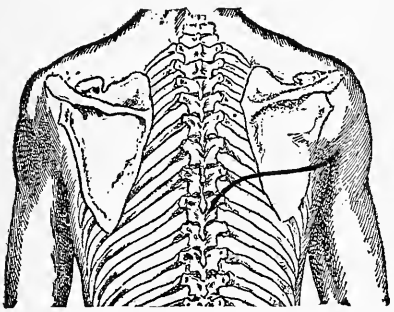


Fig. 320*a*.—Ellis's curve; moderate pleural effusion; patient in upright posture.

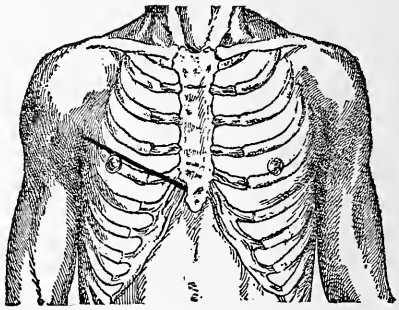


Fig. 320*b*.—Anterior line of flatness.

fered with by pleural adhesions. It is effaced when the fluid rises above the third rib. The flat percussion sound on the right side is continuous with that of the liver, from which it cannot be discriminated; on the left in the mammillary line it extends to Traube's semilunar space, the convex upper border of which becomes gradually flattened as the fluid increases. The rising and falling of the upper line of dulness, as the effusion increases or undergoes resorption, may be demonstrated by careful, light percussion and markings upon the skin at intervals of two or three days. In moderate effusions in which the lung is not confined by adhesions movable dulness may be demonstrated by marking the upper line of dulness in the anterior surface while the patient is in the erect or sitting posture, and again after some time spent in the dorsal decubitus. Massive effusions reach the clavicle and even extend to the sternal border of the opposite side. The downward dislocation of the liver or spleen may be demonstrated by linear percussion, which enables us to demonstrate the lower borders of these organs respectively. The liver is depressed in very large left effusions by reason of the dislocation of the heart toward the right, the crowding of the lung in the right pleural cavity, and the general depression of the diaphragm.

Normal Paravertebral Triangles of Relative Dulness.— Upon percussion over the spine from above downward, the resonance is progressively diminished in the lower thoracic region. This impairment of resonance also extends laterally in such a manner as to form on each side of the lower thoracic spine a narrow triangle of relative dulness, the base of which corresponds to the lower limit of normal pulmonary resonance.

Koranyi's (Grocco's) Sign; Abnormal Paravertebral Triangles of Dulness.—These triangles appear upon the sound side in pleural effusion, and may differ from the normal triangles only in respect of the degree of dulness. More commonly they differ also in extent. The procedure is as follows: (1) The borders of the effusion are determined by percussion. (2) The base of the lung upon the sound side is ascertained by percussing from above downward. (3) The degree of resonance over the spinous processes is learned by percussion also from above downward, and the point at which relative dulness begins is noted. This commonly is at the

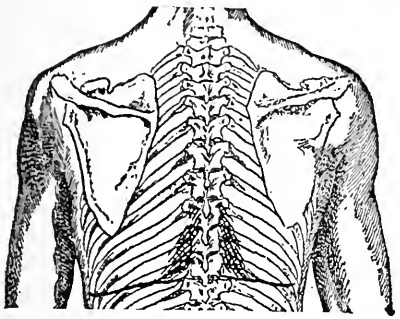


FIG. 321.—Normal paravertebral triangles.

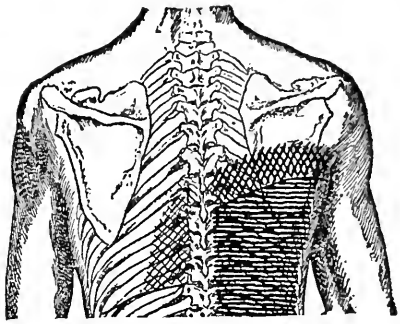


FIG. 322.—Right pleural effusion with triangle of dense paravertebral dulness on left side.

level of impaired resonance, or slightly above the level of flatness on the side of the effusion. (4) Percussion upon the sound side in a direction toward the spine in serial horizontal lines from above downward reveals a paravertebral right-angled triangle of dulness, the vertical side of which corresponds to the spine and rises to or slightly above the level of the effusion on the opposite side, the base to the lower border of the lung, while the hypothenuse extends from the apex to the outer and lowest point of dulness. The base line varies with the volume of the effusion and may reach 6 or 10 cm. in length. The triangle is usually larger in right- than in left-sided effusions. The respiratory murmur, vocal resonance, and vocal fremitus are enfeebled over this area of dulness. The phenomenon occurs alike in hydrothorax and in serofibrinous and purulent effusions. The base line is longer in purulent than in serous effusions. The triangle in free effusions disappears upon change from the upright to the recumbent posture. The explanation of the paravertebral triangles of dulness is not clear. It is probable that under normal conditions the vibrations of the lung tissue are to some extent inhibited by the bodies of the vertebræ against which it rests, and that an effusion upon the opposite side acts as a "mute" or damper and still further interferes with the vibrations of the lung in the costovertebral recess of the sound side.

Auscultation.—In the beginning of the attack friction sounds having the quality and situation of the friction signs in acute fibrinous pleurisy are heard. When the case has been under observation from the beginning, the rapid replacement of this sign by flatness is of the highest diagnostic significance. As the fluid undergoes resorption and the pleural surfaces once more come in contact, friction sounds are again heard. At this period they are grating or creaking, or fine and moist, like the crepitus of pneumonia, and are heard just above the level of flatness. As expansion of compressed vesicular tissue accompanies the process of resorption, there are crepitant râles to be heard. As the fluid accumulates, the respiratory murmur becomes at first feeble and distant. Later, while retaining its distant quality it assumes the bronchial character and may be amphoric. Cavernous respiration associated with râles, especially in children, may suggest a cavity. Over large effusions the respiratory sounds may be wholly absent. Above the level of the fluid the respiration is vesiculo-bronchial or bronchial. The vocal resonance is usually diminished or absent. In rare instances there is bronchophony. Ægophony is sometimes heard in the scapular region in medium-sized effusions. The whispered voice is better transmitted through a serous than a purulent effusion—*Bacelli's sign.*

The Heart.—The diastole is restricted by pressure. The sounds are therefore usually less distinct than normal. There is a diminished flow of blood into the arteries, which causes small pulse and a tendency to cyanosis and oliguria. Murmurs are not uncommon in the displaced heart, and a pleuropericardial friction may be detected in many cases.

Clinical Course of Serofibrinous Pleurisy.—The cases may be grouped according to the amount of fluid, which varies up to 4 litres. Less than 500 c.c. cannot be satisfactorily demonstrated in an adult. There are many cases in which the effusion does not exceed this amount and manifests itself by limited dulness at the base of the chest and immobility of the lower border of pulmonary resonance. Spontaneous arrest of the process occurs and resorption begins—*slight effusion.* In another group the fluid reaches to the level of the fourth rib in front and resorption is more tardy—*moderate effusion.* Again the upper line of flatness may reach the second rib—*large effusion;* and finally there are cases in which the outpour of the serous exudate appears to be limited only by the capacity of the pleural sac, and the whole side is distended and flat, the signs of fluid reaching to the clavicle and beyond the opposite sternal border—*massive effusion.*

In slight effusions the fever subsides, the cough ceases, and recovery takes place in the course of a week or ten days. Some impairment of resonance with feeble respiratory sounds usually persists for a longer period. In moderate and large effusions the tendency is to spontaneous resorption, but the process is slow and permanent damage to the lung results from prolonged pressure atelectasis. In this group there is commonly, but by no means invariably, a gradual subsidence of fever. The exceptional cases in which fever persists impair the usefulness of this symptom in the differential diagnosis between serofibrinous and purulent pleurisies. Massive effusions when the intrapleural pressure is extreme are usually attended

with distressing pressure symptoms. In cases in which the accumulation of fluid has been slow, the patient may experience merely a sense of weight and oppression, with shortness of breath upon exertion. Large effusions arrest the pumping function of the thoracic organs, and thereby diminish the outflow of arterial blood from, and the inflow of venous blood to, the heart, and the movement of the lymph. They show little or no tendency to undergo resorption. Serofibrinous effusions in very rare instances have perforated the lung or the chest wall. In massive effusions with great dislocation of the heart there is danger of sudden death, an accident attributed to various causes, as heart-clot, embolism of the pulmonary artery, paralysis of the heart muscle, and twists or kinking of the great vessels. The last of these explanations is purely hypothetical. Sudden œdema of the functionating lung occurs when death is not immediate.

PURULENT PLEURISY: EMPYEMA.

This designation is applied to those cases of pleural inflammation characterized by the formation of pus. When pus finds its way into the pleural sac by perforation from neighboring structures the condition is known as pyothorax.

Etiology. — Predisposing Influences. — Empyema is mostly a secondary affection. It occurs as a sequel to the infectious febrile diseases, especially scarlet fever, and is common after croupous pneumonia and bronchopneumonia and in connection with abscess and gangrene of the lung. It constitutes a rare complication of pulmonary tuberculosis, occurring in tuberculous bronchopneumonia and less frequently in consequence of pleural infection from a caseating lesion or a subpleural tuberculous abscess. Direct infection from without may also occur, as in fracture of a rib or a penetrating wound of the chest. It occurs at every age and is common in young infants, in whom a false diagnosis of pneumonia is frequently made.

Exciting Cause.—The usual organisms in their order of frequency are the pneumococcus, the ordinary pyogenic bacteria, and the tubercle bacillus. The influenza bacillus and the Bacterium coli communis have been found in rare instances. Empyema is not a stage in the course of serofibrinous pleurisy. The conversion of the serous into the purulent effusion is unusual. In very rare instances serofibrinous effusions have been infected in aspiration.

Morbid Anatomy.—The lung is compressed to an airless mass. The pleural surfaces are thickened, and are the seat of a grayish-white granular exudate. Upon the costal layer may be seen superficial erosions and sometimes the openings of fistulous tracts. The fluid has the gross and microscopical characters of ordinary pus, varying from a thin to a thick or creamy consistence. Its odor is sometimes sweetish, but in many cases, especially those following wounds or associated with gangrene, it is horribly fetid.

Symptoms.—An abrupt onset with acute symptoms is rare. The common beginning is insidious, with an intensification of the symptoms of the primary affection. Cough is neither frequent nor urgent; there is

little or no expectoration, and dyspnœa, except in large effusions, is present only upon exertion. The symptoms of sepsis, as pallor, chilliness, irregular fever, and more or less profuse sweating, are very common, especially in children. There is a high leucocytosis, 40,000 or more per cubic millimetre.

Physical Signs.—The signs elicited upon physical examination are the same as in serofibrinous pleurisy, with the following superadded: marked bulging of the affected side with obliteration, even prominence, of the intercostal spaces in the lower segment of the chest, especially in children. In many cases œdema and cyanotic discoloration of the lower part of the chest with dilatation of the venules. Whispering pectoriloquy is not heard over the effusion—*Baccelli's sign.* Distinct bronchial breathing, transmitted along the chest wall, is often heard over the effusion in young children, a sign which may lead to a false diagnosis of pneumonia. Displacement phenomena, affecting the heart, liver, and spleen, are more pronounced than in serofibrinous effusion, a fact attributed to the greater weight of the fluid, but probably due to the greater impairment in the tonicity of the tissues from the imbibition of toxin laden fluids.

VARIETIES OF PURULENT PLEURISY.

Empyema Necessitatis.—The pus by erosion of the costal pleura finds its way through an intercostal space and forms a subcutaneous, fluctuating tumor. This tumor may appear at various parts of the chest, but is usually situated anteriorly from the third to the sixth interspace. After a time if left to itself it opens, and an oblique fistulous communication with the pleural cavity is established, which continues to discharge pus for an indefinite time. When near the heart the tumor may pulsate. It is usually hemispherical and diminished in size upon full inspiration.

Pulsating Pleural Effusion.—The pulsation is synchronous with the cardiac revolution, and may be intrapleural and manifest in the lower intercostal space, in the anterolateral aspect of the chest, or show itself merely in an empyema necessitatis. The pulsation occurs in old cases, almost always upon the left side, and with one exception among the reported cases the effusion has been purulent. Various explanations have been advanced, none of which has met with general acceptance.

Encysted or Circumscribed Pleural Effusion.—The effusion is limited by pleural adhesions. The encysted fluid may vary in amount and suggest abscess of the lung, or two or more loculi may communicate with each other by narrow openings. It is sometimes serofibrinous but usually purulent. These collections may be situated between the pulmonary and costal pleuræ, especially in the posterolateral region of the chest between the base of the lung and the diaphragm, or they may be interlobar.

HEMORRHAGIC PLEURISY.

The exudate is mixed with blood. The condition is to be distinguished from hæmothorax, which arises in the absence of pleural inflammation when blood escapes into the pleural sac from traumatism, the rupture of an aneurism, or the compression of thoracic veins by a new growth.

Etiology.— Hemorrhagic effusion is of comparatively infrequent occurrence. It is encountered in pleurisy under the following conditions: in the malignant and hemorrhagic forms of the acute febrile infections; in visceral diseases associated with extensive vascular changes, as chronic nephritis and cirrhosis of the liver; in tuberculous disease, both miliary tuberculosis of the pleura and the more chronic pleural tuberculosis which accompanies chronic ulcerative phthisis; in primary and secondary malignant disease of the pleura,—carcinoma and sarcoma,—very rarely in so-called idiopathic or primary serofibrinous pleurisy, in which, however, red corpuscles are always to some extent present.

CHYLIFORM PLEURAL EFFUSIONS: HYDROPS ADIPOSUS.

The exudate has a milky appearance due to the fatty metamorphosis of endothelial and other cellular elements. The condition is not to be confounded with chylous effusion which it closely resembles. The fact is not to be overlooked that a mixture of chyliform exudates and chylous transudates may be present—as in a case recently under my care.

Etiology.— Chyliform effusions owe their peculiar appearance to, (a) the presence of cells that have undergone fatty degeneration, as in carcinoma of the pleura, tuberculous pleurisy, non-tuberculous exudates, pleurisy and abscess of the lung, and (b) to abnormal fat in the blood—lipæmia.

Diagnosis.—The nature of the effusion cannot be suspected during life unless it is withdrawn by aspiration. The fluid is yellow, whey-like, and cheesy. Upon standing there collects upon the surface a cream-like layer, showing under the microscope small globules, mostly in the form of collections of highly refractive granules with large indistinct nuclei. In the underlying fluid are leucocytes and larger cellular elements which, in consequence of differences in the amount of fat, show all possible transitional forms. (Compare this description with that of chylous effusion, p. 508.) Pseudochylous effusions have been ascribed to the presence of lecithin, and Edsall has described a non-fatty pleural effusion in which the opacity was due to altered globulins.

The Diagnosis of Pleurisy with Effusion.— DIRECT.—The diagnosis rests upon the physical signs. In large effusions the physical examination yields conclusive results. The signs may be divided into primary, or those dependent upon the presence of the fluid *per se,* and secondary, or those due to the pressure of the fluid upon adjacent organs—*displacement signs.* Among the more important of the primary signs are restricted respiratory movement, flat percussion, absence of vocal fremitus, feeble and distant breath sounds, and diminished or absent vocal resonance. The important secondary signs are displacement of the heart toward the opposite side, as shown by a visible or palpable impulse, or, in its absence, by the point of maximum intensity of the first sound: downward dislocation and immobility of the liver when the pleural effusion is right-sided; flattening of the convex upper border of Traube's semilunar space; and displacement of the spleen when the effusion is left-sided. Linear percussion shows restriction or absence of movement of the borders of the lung on the affected side, and inspection increased—vicarious—respiratory movement upon the opposite side. Difficulties arise in moderate effusions. Here the primary symptoms are usually characteristic but the valuable aid afforded

by displacement phenomena is lacking. The methods of physical diagnosis must be employed with great nicety in doubtful cases. The *S*-shaped upper line of dulness, movable dulness when present, linear percussion, flatness below and Skodaic resonance above the border line, absent or enfeebled breath sounds, and absent or enfeebled vocal resonance and vocal fremitus are significant. When several or all of these signs are present the diagnosis of effusion can be made with some confidence. In small effusions the diagnostic problem becomes more difficult and more interesting. The same signs are present, but to recognize them demands the highest skill. Finally, we have the aspirator needle which can be used in any case of doubt. There are several reasons why the aspirator should be used in exploratory puncture rather than the hypodermic syringe. The needles are longer and of larger calibre, an important matter in encysted effusions or where there is thick pus; when the exudate is serofibrinous the exploratory puncture becomes at once a therapeutic procedure and a single operation takes the place of two and when pus is present the ocular demonstration prepares the patient for the necessary later surgical operation of drainage.

The apparatus and spot selected must be sterilized, according to surgical requirements, directly before the operation. The needle should be introduced at a level in which the ordinary signs of effusion, as dulness, absent or enfeebled respiration, and absent or diminished fremitus, are well defined. As a rule, in ordinary effusions the sixth or seventh interspace in the midaxillary line, or a spot just below the angle of the scapula, may be chosen. If the puncture is made too low the needle simply penetrates the costodiaphragmatic reflexion of the pleura and may enter the liver; if too high it will be inserted into the compressed lung above the level of the effusion. The point selected for an exploratory puncture in a circumscribed lesion will be determined by the physical signs. It is an imperative rule to test the instrument with sterile water immediately before it is used. (See p. **474.**)

In small pleural effusions the employment of the röntgenological methods of examination may yield decisive positive or negative results; in large effusions they add little to the knowledge obtained by the methods of physical diagnosis.

Differential.—It is in the atypical cases that special difficulties arise.

Croupous Pneumonia.—The general rule that increased vocal fremitus occurs in pneumonic consolidation and diminished or absent vocal fremitus in effusion is subject to exceptions. In consolidation a plug or mass of tough mucus may obstruct a main bronchus and arrest the vibrations, while in effusion they may be distinctly transmitted along bands of old pleural adhesions, or in children from the opposite side along the elastic walls of the chest. The occurrence of bronchophony and bronchial respiration in certain cases adds to the difficulty. The following points are to be considered:

(1) In pleurisy, onset with moderate fever and no rigor; at most chilliness or slight chill; (2) dulness increasing to flatness at the base and posteriorly, and extending upward and forward; a peculiar sensation of inelasticity to the percussing finger; (3) vocal fremitus, enfeebled or abolished in the great majority of cases; (4) bronchial respiration, if heard at all, at the upper level of dulness or in patches; usually distant and

faint; (5) bronchophony not intense, ægophony common in the scapular region; (6) friction sounds when the case is seen early and at the upper border of dulness upon resorption of the fluid, when crepitus may be present; (7) in large effusions displacement signs; (8) sputum, mucoid when present, very rarely blood-tinged; (9) fever of irregular remittent type.

In pneumonia, (1) onset abrupt with chill, often prolonged and severe; (2) dulness rather than flatness, coextensive with the borders of a lobe or lobes; (3) vocal fremitus, marked and corresponding to the dulness, and especially when, if feeble or absent, it reappears after cough and the expectoration of tough mucoid sputum; (4) bronchial breathing most marked over area of greatest dulness and often whiffing or snoring in character; (5) bronchophony marked; ægophony rare; (6) crepitant râles, high-pitched and in "showers of crackles" diffused over an area of dulness and disappearing when bronchial breathing becomes intense; (7) displacement phenomena absent; (8) rusty or prune-juice sputum the rule; (9) high temperature of typical range, self-limited course, and critical defervescence. Pleurisy with effusion is frequently associated with croupous pneumonia and bronchopneumonia.

Pericardial Effusion.—When large this condition may simulate left-sided pleural effusion. The outline of the area of dulness anteriorly, its convexity to the right of the sternum, Skodaic resonance at the base and in the axillary region, absence of cardiac impulse on the right, and a degree of dyspnœa and cardiac feebleness not seen in moderate pleural effusions are of diagnostic importance.

Hydrothorax.—When unilateral, this condition cannot always be differentiated from serofibrinous pleurisy by the ordinary methods of physical examination. It occurs in heart disease with great enlargement or dilatation. The diagnosis rests upon concomitant conditions and character of the fluid.

Intrathoracic Tumors.—New growths of the lung, pleura, and mediastinum may be mistaken for pleural effusion. The situation of the dulness and its irregular outline, the signs of marked compression of the large venous trunks, important nerves, and hollow organs, as the trachea, bronchi, and œsophagus, indications of malignant disease in other parts of the body, and enlargement of superficial lymph-nodes should prevent this error. Intrathoracic tumors are very often complicated by pleural effusion.

Aneurism.—Pulsating empyema necessitatis may suggest aortic aneurism. The location of the tumor, usually at the base of the chest, the absence of murmurs, diastolic shock, and tracheal tugging, and the fact that on deep inspiration the tumor diminishes in size and tension are against the diagnosis of aneurism. A fine exploratory needle may be introduced.

Extrapleural Abscess.—This rare condition is to be differentiated from pleural effusion by the absence of the signs of compression of the lungs and the displacement of adjacent organs. When such an abscess is opened pneumothorax does not occur and a probe does not enter the pleural cavity.

Subphrenic Abscess.—This condition may suggest a moderate pleural effusion, from which it may be differentiated by the persistence of the respiratory movement of the lower border of the lung, the presence of food particles in the aspirated fluid when the condition is due to gastric ulcer perihepatic friction, and the absence of pneumothorax upon puncture.

Tumors of the Liver.—Abscess, hydatid cyst, and carcinoma in the right lobe of the liver may displace the diaphragm upward, compress the lung, and cause dulness and feeble respiratory murmur. If large they may also dislocate the cardiac impulse slightly to the left. The diagnosis of hepatic enlargement rests upon the retention of the respiratory movement of the lower border of the lung, friction sounds over the area of dulness, and demonstrable convexity of the upper line of dulness. Exploratory puncture may be performed.

Perforation of the diaphragm may, when adhesions to the liver have taken place, cause a condition not to be differentiated clinically from hepatic abscess, unless the case has been observed from its onset, or pus which is characteristic of hepatic abscess is expectorated or obtained by operation. Röntgenological methods may be imperative.

The Diagnosis of the Character of the Effusion.—This may be readily settled by the use of the aspirator needle. With this means at our disposal the clinical symptoms, which are somewhat uncertain, assume secondary importance. A serofibrinous effusion is suggested by comparatively mild onset, the absence of the evidence of previous disease, the transmission of the whispered voice, and in general a mild course characterized merely by malnutrition, anæmia, and dyspnœa upon exertion. That the effusion is purulent is rendered probable by the presence of pneumonia, influenza, sepsis or phthisis, irregular chills, high fever and copious sweating, non-transmission of the whispered voice, œdema and cyanosis of the lower portion of the affected side, and a high leucocytosis. But pus may be present in default of several of these symptoms, on the one hand, and, on the other, severe septic phenomena may accompany a moderate, even a small circumscribed serous effusion. Hemorrhagic and chyliform fluids can be recognized only when withdrawn.

The Diagnosis of the Pathological Process.—The examination of the fluid is of great service both as regards diagnosis and prognosis. A majority of serofibrinous effusions are of tuberculous origin. Tuberculous foci may or may not be present in the lungs. The methods of examination comprise microscopy, which may be employed at the bedside, and cytodiagnosis, animal inoculation, and culture methods, which are available only in the laboratory. Lymphocytes generally predominate in tuberculosis effusions; a polynuclear leucocyte preponderance suggests acute infection, and a large number of endothelial cells is found in mechanical effusion or transudate. Inoculation methods, with small amounts of the fluid, as usually practiced are negative, but when larger quantities, as 15 c.c., are used, the result has confirmed the clinical and pathological findings in regard to the preponderance of tuberculous cases in serofibrinous effusions. If actual fragments of cancerous tissue are present the diagnosis is positive. Bacteria are present in small numbers in clear exudates. In purulent exudates they are present in great numbers, sometimes a single variety, sometimes several varieties. Streptococci are most commonly present. The infection may be direct from the lung, as in bronchopneumonia or streptococcus pneumonia, or from distant foci. Less common is pneumococcus infection, which is usually secondary, exceptionally primary.

The Prognostic Value of the Bacteriological Examination of the Fluid.—A sterile fluid usually may be regarded as of tuberculous origin. The presence of the pneumococcus is relatively favorable, since the cases generally run a satisfactory course and recovery may take place after a single aspiration. Streptococcus pleurisy is the most unfavorable of all forms. It is frequently associated with general septicæmia and leads up to the fatal issue. The mixed infections are of unfavorable import.

The Prognosis of Purulent Pleural Effusions.—Empyema is an essentially chronic disease. If neglected the outlook as to recovery is extremely unfavorable, and when spontaneous recovery occurs it is only partial. Early and efficient drainage is followed by a large proportion of satisfactory recoveries. The most unfavorable cases are those which arise in the course of general streptococcus infection. Untreated cases may terminate: 1. In small empyemata, by gradual resorption of the fluid and the deposition of lime salts. 2. By the discharge of the pus through the lung, more commonly after the establishment of a bronchopulmonary fistula, very rarely by soakage without the formation of a demonstrable fistula. In the former case pneumothorax almost always occurs, in the latter probably never. If sudden rupture occurs life may be destroyed by suffocation. 3. By the perforation of the costal or diaphragmatic pleura and the formation of empyema necessitatis which, though usually in the anterior surface of the chest, may be at any point, including the lumbar region and the iliac fossa, where it simulates a lumbar or psoas abscess. Under these circumstances there is usually permanent atelectasis of the lung with fibroid changes, great pleural thickening, and contraction and deformity of the chest. A fair degree of health may be maintained for a varying period, but if the patient survive there is clubbing of the finger-tips, amyloid disease develops, and ultimately tuberculosis in a large proportion of the cases.

Morbid States Characterized by the Transudation of Serum or Chyle, or the Eruption of Pus, Blood, or Air Into the Pleural Sac.

(a) HYDROTHORAX.

Definition.—The accumulation of simple non-inflammatory fluid in the pleural cavities. It occurs as a secondary affection in many diseases, chiefly those attended by dropsy.

Etiology.—The primary disease may involve the kidneys, the heart, or the blood. There is usually more or less anasarca, exceptionally merely slight œdema of the feet. Hydrothorax is in many cases the precursor of death. In disease of the kidneys it is commonly bilateral, the effusion being greater on one side, usually the right. In chronic valvular disease with hypertrophy and dilatation, the effusion is always more marked and sometimes solely upon the right side, and it promptly returns after repeated aspiration. The larger right-sided effusion of cardiac disease has been ascribed to pressure upon the azygos veins, but it is probably due to the larger space in the left thorax occupied by the enlarged heart. Extensive old pleural adhesions may prevent accumulation upon

one side. The pleural membranes are not the seat of a fibrinous exudate, being smooth and glistening. The fluid is clear and free from fibrin flocculi. It is usually moderate in amount. The symptoms are dyspnœa, often amounting to orthopnœa, and an aggravation of those due to the primary disease. The physical signs are those of pleural effusion.

Diagnosis.—The condition may be differentiated from serofibrinous pleurisy by the nature of the primary disease, the absence of fever, of displacement symptoms, of friction sounds, and the relatively prompt change in the line of dulness with change of posture.

(b) CHYLOUS PLEURAL EFFUSION—HYDROPS CHYLOSUS.

Definition.—An accumulation of chyle from the thoracic duct or the lacteals by transudation or direct discharge into the pleural sac.

Etiology.—The special causes of chylous effusion into the pleural sac are, (a) conditions leading to an escape of chyle, as external violence, disease or occlusion of the chyliferous vessels, carcinoma of the pleura, occlusion of the left subclavian vein, compression of the duct by a tumor, malignant lymphoma, disease of lymphatic vessels, sclerosis, lymphangiectasis, and filariasis, and (b) the discharge of a chylous ascites into the pleural cavity by way of the lymph spaces.

Symptoms.—The symptoms and physical signs do not differ in any particular from those of a pleural effusion.

Diagnosis.—There are no means by which the nature of the fluid can be determined *intra vitam* except by the withdrawal of a portion of it. In many of the reported cases its presence was first recognized at the autopsy. The fluid bears the closest resemblance to milk, is literally milk-like. It is opaque white in color, with a faint yellowish or creamy shade, slightly alkaline, and of a specific gravity of about 1,017. Microscopically there are seen great numbers of minute, dust-like granules in active, molecular movement, a few larger fatty bodies scattered separately or in groups, a few leucocytes, larger cells containing distinct, highly refracted granules, and a very few erythrocytes. Shaken with ether in a test-tube after the addition of a few drops of potassium hydroxide, the fluid becomes transparent and almost colorless. Upon the addition of osmic acid it becomes black in color. The morphological elements are almost exclusively leucocytes and, in great preponderance, lymphocytes. The fluid is sterile. The foregoing characters serve to distinguish chylous effusion—transudates—from chyliform effusions—exudates (see p. 503). The presence of grape-sugar is without diagnostic importance, since the fact has recently been established that this substance may frequently be demonstrated in ordinary serous transudates and exudates, and may therefore be expected in chyliform exudates.

(c) PYOTHORAX.

The sudden rupture of a hepatic, subphrenic, mediastinal, or pulmonary abscess into the pleural sac may take place. This accident is usually prevented by more or less extensive pleural adhesions. When it occurs, general infection of the pleura immediately follows with the

conversion of pyothorax into purulent pleurisy. Communication with the bronchi or with a subphrenic pneumothorax will give rise to the association of air with the pus—pyopneumothorax.

(d) HÆMOTHORAX.

Hemorrhage into the pleural cavity results from trauma, the rupture of an aneurism, the pressure of a tumor upon the thoracic veins, and in rare instances from pulmonary gangrene. The sudden manifestation of the symptoms of internal hemorrhage, pallor, collapse, small, thready pulse, coupled with the physical signs of pleural effusion justify a provisional diagnosis. The withdrawal of blood upon exploratory puncture renders the diagnosis positive.

(e) PNEUMOTHORAX.

Hydropneumothorax; Hæmopneumothorax; Pyopneumothorax.

Definition.—Air in the pleural cavity. This condition is extremely rare. Infection of the pleura takes place in almost every instance, and in the course of a short time the air is associated with fluid—*hydropneumothorax*, *hæmopneumothorax*, or *pyopneumothorax*.

There exists in the normal pleural cavity a negative pressure, by reason of which the lung fills the chest in a state of vital tension. When, through any communication with the external atmosphere, the tension is relieved, the distended lung collapses to the limits of its inherent elasticity, and a volume of air, equivalent to the differences in the mass of the lung under normal distention and under balanced intrapulmonary and intrapleural pressure, enters the pleural sac—*pneumothorax*. This balance is, however, maintained only in the case of the communication remaining freely open as in some external wounds or perforation through consolidated lung tissue. Under other circumstances a valvular action is established, particularly in the perforation through the lung, and the intrapleural pressure gradually becomes positive. While the balance is maintained, the mediastinum is *drawn* toward the opposite side, and the diaphragm somewhat depressed; when the pressure becomes positive, displacement phenomena become more marked, the mediastinum is *pushed* further toward the sound side, and the diaphragm pushed downward.

Etiology. — Pneumothorax is caused by: 1. Perforating wounds of the pleura: (a) through the chest wall, as in the case of stabs and gunshot injuries, aspiration and other surgical operations; (b) internal trauma, as when sharp or pointed foreign bodies are swallowed, or an emphysematous lung or one tied down by local adhesions is torn in violent efforts at lifting or in paroxysms of cough. The accident may even occur in the absence of straining. The air may gradually undergo resorption. More commonly pleurisy with effusion follows. 2. Perforation of the pleura by ulceration or necrosis: (a) from without, as in (i) diseases of the lung: (*a*) tuberculosis, by far the most common cause, the perforating lesion being the softening of a caseous mass or the rupture of a rapidly forming cavity before limiting pleural adhesions have taken place. (*β*)

Necrosis of lung tissue in septic conditions, as septic bronchopneumonia, gangrene, and very rarely infarctions. (ii) Malignant disease of the œsophagus. (iii) Infradiaphragmatic lesions, as (*α*) subphrenic abscess, (*β*) abscess of the liver, (*γ*) malignant disease of the stomach or colon. (b) From within, as in empyema, with the formation of a bronchopulmonary or pleurobronchial fistula. 3. As the result of the development in pleural exudates of the gas-producing bacillus—B. aërogenes capsulatus of Welch.

In rare cases pneumothorax is double, and recurrent cases have been reported. The condition is common in adults, exceptional in children.

Morbid Anatomy.—The air space is usually large, the lung compressed and carnified, the pleura inflamed, and serous or purulent effusion present. The confined air may escape through a cannula with a whistling sound and force enough to blow out a candle.

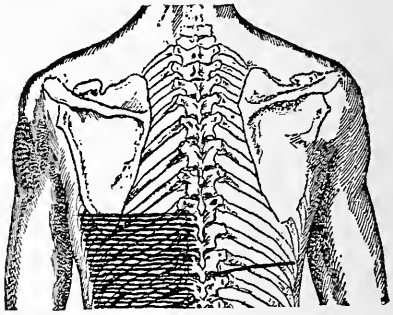


Fig. 323.—Horizontal line of surface of effusion in pyopneumothorax; patient in the upright position. This line shifts with change of posture.

Symptoms.—The occurrence of pneumothorax is usually attended by sudden pain in the side, distressing shortness of breath, slight cyanosis, and feeble pulse. In old tuberculous cases it may occur insidiously.

Physical Signs.—The results of physical examination are characteristic. Inspection.—The affected side is enlarged, the intercostal spaces bulge, and the respiratory excursus is greatly diminished. The impulse of the heart is displaced toward the opposite side. Palpation.—Vocal fremitus is absent or greatly diminished. Percussion.—The signs depend upon the degree of intrapleural tension and the amount of fluid present. When tension is moderate the physical conditions necessary to the production of tympanitic resonance are present, and as these conditions vary the quality of tympany changes from clear, high-pitched hyperresonance to the flat, woodeny tympany of Skodaic resonance. When tension is extreme the physical conditions underlying tympany no longer exist, and the percussion sound is muffled and dull—a fact of great importance in diagnosis. There is flatness at the base due to effused fluid, its upper horizontal line indicating the height to which the effusion rises and changing with change of posture—*movable dulness*. Auscultation.—The breath sounds are feeble and distant and have the amphoric quality. They are in strong contrast with the loud puerile vesicular murmur of the sound side. The voice has also a peculiar, amphoric quality. The ringing musical râle known as *metallic tinkling* or *gutta cadens* is heard upon deep breathing or coughing. The coin test is also present and of positive diagnostic value. Finally, the swash of the free fluid within the pleural cavity upon energetic sudden changes of the patient's body—*Hippocratic succussion*—may often be heard at a distance, or even by the patient himself. In that form of pneumothorax in which there is free communication with a bronchus through consolidated lung, the bulging of the intercostal spaces and dis-

placement signs are less marked, coarse, gurgling râles are observed at times, and there is the occasional expectoration of a thin, purulent fluid. In rare instances of left-sided pneumothorax the heart sounds may have a metallic echoing quality.

Diagnosis.—Direct.—The signs are characteristic and the diagnosis, even when in consequence of old adhesions the air space is limited, may be made with confidence.

Differential.—The following conditions may give rise to uncertainty: *Cirrhosis of the Left Lung.*—The high position of the diaphragm with a dilated stomach yields tympany in the lower part of the left chest, amphoric sounds, and sometimes gastric succussion. The various sounds are little influenced by respiration. Percussion when the stomach is filled with fluid and the fact that the impulse of the heart is displaced toward the left are conclusive. *Diaphragmatic Hernia.*—This condition when congenital may be misleading. After a crushing accident the metallic sounds are related to peristaltic rather than respiratory movements, and the difficulty in passing the tube at the cardiac orifice, owing to the dislocation of the stomach, is suggestive. *Pyopneumothorax Subphrenicus of Leyden.*—The anamnesis is important—symptoms of gastric or duodenal ulcer, chronic intestinal disease, hepatic or splenic abscess usually precede this condition. Cough and sputum are not commonly present; the heart is slightly displaced, the liver much lowered. The lower border of the lung rises and falls, as shown by percussion and auscultation, upon deep respiratory efforts, and movable dulness cannot be made out. *Large Intrapulmonary Cavities.*—Two conditions are to be considered: first, the breaking down of the greater part of a lung—a very rare event—in which the physical arrangement closely resembles ordinary pneumothorax; and second, the cavities resulting from pulmonary abscess, gangrene, or bronchiectasis, or the ordinary cavities of phthisis, which may simulate circumscribed pneumothorax, which is likewise of extremely rare occurrence. In the first of these conditions the amphoric quality of the respiratory and voice sounds may be intense, but the succussion splash, the coin sound, and displacement phenomena are absent. In extremely rare cases, however, coin percussion may yield the bell-like resonance over a cavity. Smaller cavities may be differentiated from circumscribed pneumothorax by the presence or increase of the vocal fremitus, absence of chest distention, sinking of the intercostal spaces over the cavity, and changes in the physical signs, upon percussion and auscultation, after severe cough with copious expectoration. The location of the cavity is without diagnostic importance, since sacculated pneumothorax may occur at the apex, while the cavities following abscess and gangrene are usually in the lower lobe, those of bronchiectasis may occupy any portion of the lung, and those of phthisis, while usually apical, are sometimes situated at the base.

Prognosis.—Spontaneous pneumothorax, occurring upon effort in a person suffering from emphysema or with local pleural adhesion, frequently terminates in recovery with resorption of the air. The traumatic and surgical cases also do well. There is a group of tuberculous cases in which the occurrence of pneumothorax appears to arrest the progress of the disease. There are chronic cases of open pneumothorax, the fistula

being either pleurobronchial or external, which last for years, the patients being able to go about and attend to their affairs. In pneumothorax acutissimus death may take place within an hour or in the course of the first day, though the catastrophe may be averted by the use of the trocar and cannula. As a rule, the cases occurring in tuberculosis die in a few days or weeks.

Masked Pneumothorax.—This term has been applied to cases in which the symptoms of pneumothorax, namely, intense pleural pain, dyspnœa, pressure phenomena, and displacement of the mediastinal organs and diaphragm, have suddenly occurred in the course of advanced tuberculosis in the absence of the usual signs of pneumothorax upon auscultation and percussion. In the course of some days these signs gradually appear, and a circumscribed pneumothorax may be demonstrated. They are at first obscured by the deep situation of the collection of extrapulmonary air, which has escaped by way of an opening into the mediastinum, an interlobar space, or a space between the base of the lung and the diaphragm, and is retained by previously formed pleural adhesions. Deep-seated circumscribed pneumothorax is sometimes encountered post mortem in cases in which neither the signs nor symptoms have been observed during life.

XI.

THE DIAGNOSIS OF DISEASES OF THE KIDNEYS.

I. ANATOMICAL ANOMALIES OF THE KIDNEYS.

The kidneys may be displaced, with or without deformity; the displacement may be congenital or acquired. They may vary in number: congenital absence of one or both kidneys, supernumerary kidneys, atrophy of one kidney. They may be anomalous in form: general departures from type, as lobulation; hypertrophy of one or both organs, and fusion —horse-shoe kidney, sigmoid kidney, disk-shaped kidney. Finally, there may be variations in the blood-vessels, pelvis, and ureters.

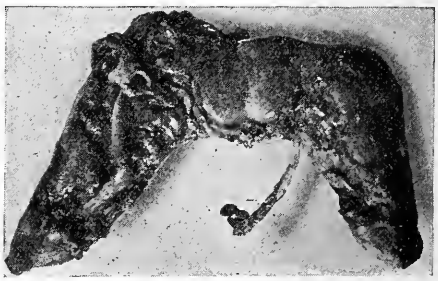


Fig. 324.—"Horse-shoe" kidney —German Hospital.

Of these abnormal conditions the hypertrophied kidney can be diagnosticated only when the affected organ is movable and is recognized upon palpation through the abdominal wall; the horse-shoe kidney only when it has descended to a position just above the promontory of the sacrum and can be felt through thin abdominal walls as a prevertebral tumor with a non-expansile pulsation communicated from the underlying aorta, upon which it in part rests; a single kidney may be suspected when tympanitic percussion resonance is constantly elicited in one lumbar region and no movable kidney is palpable, or when, after an attack of

renal colic with impaction, complete anuria and ultimately fatal uræmia occur. In rare cases the impaction of a calculus upon one side may be followed by anuria when both kidneys are present. Other anomalies cannot be recognized during life.

II. MOVABLE KIDNEY.

Ren Mobilis; Palpable Kidney; Floating or Wandering Kidney; Nephroptosis.

Etiology.—The condition may be congenital, the kidney being surrounded by peritoneum which forms a mesonephron. Far more commonly it is acquired. It is probable that congenital defects in the mechanism of attachment are at fault in all cases. Wasting of the perirenal fat is a factor. Movable kidney has been observed at all ages, but is most usual in middle life. It is more common in women than men in the proportion of 7 to 1—a fact attributed to compression of the base of the chest by the corset and the change in the position of the uterus and the relaxation of the abdominal wall after repeated child-bearing. It occurs, however, in women who have never borne children. It is mostly unilateral, several times as often on the right side as on the left, and occasionally double. The greater frequency on the right side is attributed to the relation of the right kidney to the liver and the respiratory movement communicated to it by the latter organ. In the anamnesis there is sometimes an antecedent history of injury or strain. The kidney undergoes dislocation together with the other abdominal viscera in Glénard's disease—*enteroptosis.*

Symptoms.—In a large proportion of the cases there are no definite or characteristic symptoms. Neurasthenic and gastro-intestinal symptoms are common. Constipation is frequent and fecal obstruction may occur. Dragging pains in the lumbar region, especially upon prolonged standing, are observed. Neuralgic pains in the abdomen occur. The tumor, which is often accidentally discovered by the patient, is not tender upon gentle pressure, but when firmly compressed there is a dull, sickening pain. *Dietl's Crises.*—In some cases of floating kidney there are paroxysmal attacks, characterized by abdominal pain, nausea, and vomiting, with chills, fever, and collapse. These attacks have been mistaken for renal colic, acute intestinal disease, and appendicitis, but the kidney may be felt and is tender, swollen, and less freely movable than usual. The urine during the attack may contain uric acid or calcium oxalates in excess, and intermittent hæmaturia may occur. These paroxysms have been ascribed to torsion of the renal vessels. Intermittent hydronephrosis sometimes occurs. The nervous symptoms of movable kidney are important. In women hysterical manifestations, in men hypochondriasis are common. Such patients are very susceptible to suggestion, and their sufferings are often much increased when the diagnosis is communicated to them. In other cases a plain statement of the cause of the trouble is followed by relief.

Physical Examination.—The patient should be placed upon his back, with the abdominal muscles relaxed. In well-marked cases the tumor is plainly visible and palpable in the erect posture. Ordinarily, upon bimanual palpation in the dorsal decubitus, one hand being placed in the lumbar

region, the other in the hypochondrium, with gentle manipulation during full respiratory movements, the kidney, if movable, may be recognized by the fingers upon the abdomen as an oval, smooth mass. By this manœuvre various degrees of mobility may be determined. (1) PALPABLE KIDNEY.—The lower end of the organ may be felt upon deep palpation just below the edge of the ribs in the nipple line—a condition of little or no clinical import. (2) MOVABLE KIDNEY.—Upon deep inspiration the fingers upon the abdomen may, if there is little abdominal fat, be pressed over the upper end of the kidney, which can be thus fixed for the time but has no wider excursion—a degree of dislocation the importance of which is frequently overrated. (3) FLOATING OR WANDERING KIDNEY.—The organ may be felt as an oval, smooth, solid tumor, having the size and contour of the kidney. In some instances the hilum and pulsating renal artery can be recognized. This tumor is freely movable, and sometimes lies just above Poupart's ligament, or may by gentle pressure be displaced to the median line or beyond it. In different postures the wandering kidney changes its position, falling forward in the knee-elbow position, and away from the abdominal wall in the dorsal decubitus, when it is often possible to slip it upward into its normal place. To this degree of displacement belong the more distressing symptoms of *ren mobilis*. Dilatation and downward displacement of the stomach can be demonstrated in a large proportion of the cases, especially in women.

Diagnosis.—DIRECT.—A positive diagnosis can usually be made in palpable and movable kidney by the position of the smooth, rounded, firm tumor, which descends with deep inspiration and can be made to disappear by pressure upward and backward, particularly when, as sometimes happens upon repeated examination, flattening or tympanitic resonance is found in the renal region upon the same side. Floating kidney rarely presents difficulty in diagnosis. See examination by Röntgen rays.

DIFFERENTIAL.—*Tumor of the Gall-bladder.*—The mass presents at the border of the ribs, has the respiratory movement of the liver, cannot be grasped from above, and when forced backward immediately returns to its former position. Its movement is less extensive than in floating kidney and is, roughly speaking, in the arc of a circle having its centre in the normal position of the gall-bladder. Furthermore, in tumors of the gall-bladder the upper margin reaches and is continuous with the liver. Other tumors of the liver and tumors of the bowel are fixed and do not present the characteristic contour of the kidney. *Movable Spleen.*—Any doubt as to whether a movable tumor upon the left side is the kidney or spleen is at once settled by the shape of the tumor and the presence or absence of the normal area of dulness in the splenic region. *Tumor of the Pylorus.*—Carcinoma in this region may be freely movable. Under such circumstances the shape of the tumor, its relation to the stomach, filled and emptied by means of the stomach tube, dulness in both renal regions, and the prominence of gastric symptoms are of diagnostic value. *Ovarian Cysts.*—The facts that the tumor arises from the pelvis, that its outline is round or globular, that it is elastic rather than firm, and that it cannot be made to disappear into the normal position of the kidney, readily settle any doubt as to the differential diagnosis.

Prognosis.—The outlook as to permanent fixation is less hopeful than as to relief by the adjustments that follow improvement in the general health. Nephropexy and nephrorrhaphy, with and without decapsulation, have many successes and many failures to their credit. Relief in many cases may be obtained by a suitable belt and pad and treatment of the neurasthenia.

RENAL DISEASES.

The pathological conditions of the kidneys comprised under the general term Bright's disease cannot be satisfactorily classified, either from the clinical or the pathological standpoint. Still less can the clinical varieties be closely coördinated with the post-mortem findings. Clinically the cases may be grouped as acute and chronic; etiologically as toxic or infectious; anatomically they are all diffuse; that is to say, epithelial, vascular, and intertubular tissues are involved, but, since the changes in these structures vary in degree, parenchymatous, glomerular, and interstitial forms are recognized, according as one or the other of these groups of tissues is particularly affected.

Bright's Disease.—This unfortunate designation is still sometimes applied to conditions characterized by renal symptoms and attended by albuminuria.

Nephrosis.—The nephroses comprise the toxic, degenerative forms of renal disease, in which the tubules are involved without marked glomerular implication. They include febrile albuminuria, the condition in sublimate, chrome and other intoxications, in pregnancy and the amyloid kidney (*q.v.*).

Nephritis.—Inflammation of the kidney. The forms are acute, subacute, and chronic. The nephritides comprise diffuse glomerulonephritis, acute and chronic, interstitial nephritis and the specific forms of inflammation of the kidneys caused by tuberculosis and syphilis (*q.v.*).

III. CIRCULATORY DERANGEMENTS.

Theoretically anæmia and congestion occur.

(a) **Renal Anæmia.**—No positive diagnosis of anæmia of the kidneys can be made.

(b) **Congestion of the Kidneys.**—Active Congestion.—*Etiology.*—Certain drugs as the terebinthinates and cantharides, when taken in overdoses, are accredited with causing congestion of the kidneys. Exposure to damp and cold, various poisons and irritants have the same effect. Active hyperæmia is characteristic of the onset of acute nephritis, from which it cannot be clinically differentiated. Post mortem the kidney is large, dark, and soft, and upon section drips blood. The condition is typical in postscarlatinal nephritis. The urine is scanty, densely albuminous, and contains red blood-corpuscles and tube-casts.

Chronic Passive Congestion.—Stasis Kidney.—*Etiology.*—The hyperæmia results from the transference of blood-pressure from the arterial to the venous side of the circulation, which occurs in cardiac disease and

emphysema, and locally from pressure upon the renal veins by the pregnant uterus, abdominal tumors, and large ascites. The condition found post mortem is known as cyanotic induration. It is a chronic tubular degeneration. The urine is diminished, dark red in color, of high specific gravity, and contains albumin in moderate amount, with hyaline tube-casts. A few red blood-corpuscles may be present in the sediment. The line between congestion and nephritis cannot always be drawn at the bedside. Hyaline casts only, moderate albumin, isolated red corpuscles, total absence of uræmic symptoms, cynosis rather than pallor, and improvement upon the administration of digitalis suggest, in a heart case, the diagnosis of congestion rather than nephritis. *Prognosis.*—The causal conditions in chronic hyperæmia of the kidneys are such as to render the prognosis unfavorable.

(c) **Hemorrhagic Infarct of the Kidney.**— *Etiology.*—Embolism of renal arteries occurs in valvular disease, endarteritis, and traumatism involving the renal artery. *Symptoms.*—Sudden pain in the region of the kidney upon one side, with corresponding tenderness upon pressure, and hæmaturia constitute the symptoms in well-marked cases. These symptoms are all transient, the pain and tenderness subsiding in the course of a day or two, the blood disappearing from the urine in three or four days, and the albumin a short time later. In the majority of instances in which infarcts are found post mortem no clinical symptoms have been noted. *Diagnosis.*—Hemorrhagic infarct of the kidney cannot, as a rule, be diagnosticated during life. When the above symptoms occur in a patient in whom the etiological factors are present, or in whom embolic processes elsewhere can be demonstrated, the diagnosis is positive. *Prognosis.*—The outlook is that of the underlying morbid conditions. Old infarcts of the kidneys are often found in post-mortem examinations.

IV. URÆMIA.

Definition.— A toxæmia developing in the course of acute or chronic nephritis and other conditions characterized by deficient urinary secretion or complete anuria, and manifested by irregularly associated nervous and gastro-intestinal symptoms.

Various hypotheses have been advanced regarding the pathology of uræmia, among which the following are important: 1. That it is due to the accumulation of excrementitious substances normally eliminated by the kidneys, especially urea, salts, and nitrogenous bodies. 2. That it is caused by toxins evolved in the course of abnormal tissue metabolism, of the nature of which nothing positive is known. Uræmia has been attributed to derangements of a hypothetical internal secretion of the kidney. 3. That the nervous symptoms are largely due to local cerebral œdema.

Symptoms.—Uræmia may be of every grade of intensity and of the most variable duration. Latent, acute, and chronic forms are therefore described.

The Latent Form.—This form has been especially studied in cases of complete anuria. The patient may suffer very little inconvenience. Preliminary headache and the alternation of convulsions and coma seen in acute

uræmia are often absent. The mind remains clear, the pupils are contracted, muscular twitchings and vomiting occur. The temperature is subnormal.

The Acute Form.—The onset is preceded by headache, mental confusion, dulness, and drowsiness. The attack begins abruptly with vomiting and diarrhœa, or convulsions alternating with or followed by coma, or coma may develop in the absence of convulsions. Such an attack very often occurs in persons in whom no previous indications of nephritis have been observed. Fever of irregular type is frequently present, and may be, in acute nephritis, a manifestation of the underlying disease or symptomatic of some complication, as an intercurrent inflammatory or infectious process, itself the cause of the uræmia, or the fever may be part of the uræmic symptom-complex.

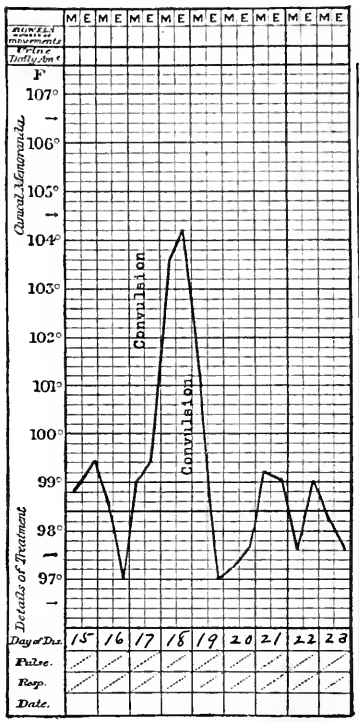


Fig. 325.—Chronic parenchymatous nephritis. Uræmia; convulsions.

The Chronic Form.—The patient may go about and in a way attend to his affairs. He suffers, however, from headache, vertigo, confusion, drowsiness, and pruritus, and very often has transient muscular twitchings. Dyspnœa, which may be continuous or paroxysmal, and is frequently nocturnal only, is a common symptom in chronic uræmia. It is often regarded as asthma. The respiration is sometimes Cheyne-Stokes in type. Itching, numbness, and cramps in the calves of the legs also occur. Local palsies, hemiplegias, and monoplegias occur, and are frequently transient. The psychoses of chronic uræmia are important. They very often occur in persons not known to have nephritis. Mania and delusional insanity are common. Delusions of persecution, suicidal tendencies, and melancholia occur. The alienist may be in doubt whether an insane person has nephritis, or a patient suffering from nephritis has an uræmic psychosis—an uncertainty which emphasizes the artificial character of nosological classifications.

The convulsions of uræmia may occur abruptly or after a spell of headache and restlessness. They closely resemble the epileptic seizure, though the epileptic cry is said not to occur. The repetition of the general convulsion with unconsciousness in the intervals may suggest "status epilepticus." Jacksonian epilepsy may occur. The temperature sinks as a rule after the attack. Uræmic amaurosis may occur after a convulsive attack, or in the absence of convulsions. It may pass off in the course of a few days. The ophthalmoscopic findings are negative. Uræmic deafness of the same fleeting character has also been observed.

Persons suffering from chronic uræmia frequently have no appetite and a foul tongue and breath. The stomatitis sometimes present has no special characters. The blood-pressure is almost always high.

In grave cases of acute uræmia a frost-like efflorescence of urea has sometimes been observed upon the skin. Acute inflammations of the serous membranes, endocarditis, pericarditis, pleurisy, peritonitis, and, much more rarely, meningitis occur as terminal events in patients suffering from conditions in which chronic uræmia has developed.

Diagnosis.—DIRECT.—The diagnosis of uræmia depends upon the association of nephritis and nervous symptoms of more or less irregular character and combination. In cases of anuria from any cause a direct diagnosis is justified. In other cases an examination of the urine yields definite data. The nervous symptoms are often such that a diagnosis by exclusion becomes necessary. The copious vomitus in some cases may have the odor of ammonia, since the urea in the gastric contents may have undergone the change into ammonium carbonate. The determination of the urea output in the urine cannot be relied upon as indicating the approach of uræmic symptoms.

The diagnosis of the latent uræmia of Roberts rests upon the association of certain of the milder symptoms of the condition with more or less complete anuria. In acute fully developed uræmia with vomiting, convulsions, coma, amaurosis, and stertorous or Cheyne-Stokes respiration, the symptom-complex is so characteristic that errors in diagnosis seldom occur. If some of these symptoms suddenly develop as the result of intoxications or severe infectious processes, in persons not suffering from nephritis, the presence of albumin in relatively small amounts and hyaline casts only, without red blood-corpuscles or other tube-casts, particularly if the specific gravity of the urine be not abnormally low, is of diagnostic value. Nor is it always easy to recognize the nausea, vomiting, and diarrhœa of the gastro-intestinal form as uræmic. Chronic uræmia is even more difficult of recognition. Asthma-like attacks with shortness of breath, especially at night, may lead to an incorrect diagnosis in cases in which the nervous symptoms are slight and ill-defined. The acute attack very often occurs in the course of the chronic condition. The pupils are inconstant. They may be dilated or normal. The presence of albuminuric retinitis may be of positive diagnostic significance.

DIFFERENTIAL.—1. CEREBRAL DISEASE.—(a) The uræmic attack, with sudden loss of consciousness, and hemiplegia, especially when these symptoms are associated with convulsions, may present the clinical picture of *apoplexy* from cerebral hemorrhage or thrombosis. In favor of the latter diagnosis are the abruptness of the onset, the completeness of the loss of power, conjugate deviation, and persistence of the symptoms. It is characteristic of the nervous symptoms of uræmia that they are incomplete and transitory. (b) *Meningitis.*—Sudden coma following headache and vomiting, without localizing phenomena but attended by albuminuria, may present great difficulties in diagnosis. The results of spinal puncture are important. Stiffness of the neck, paralysis of cerebral nerves, retraction of the abdomen, and Kernig's sign occur in meningitis. (c) *Tumors.*—

The symptoms of coarse lesions of the brain may suggest uræmia. Jacksonian and general convulsions, vomiting, headache, vertigo, and hemiplegia and monoplegia occur in both conditions. But in anatomical lesions the symptoms are chronic and usually though not always progressive, while in uræmia characterized by such symptoms the attack is sudden and frequently transitory. 2. Severe Infections.—There are cases of uræmia in which stupor, a dry tongue, rapid, feeble pulse, muscular twitching, and fever persist for weeks, and the appearance of the patient suggests an acute specific disease with secondary infection. These cases may resemble: (a) *Enteric Fever.*—The differentiation from uræmia depends upon the presence of a pulse relatively slow, as compared with the rise of temperature, splenic tumor, rose rash, a temperature range conforming to type, and a positive Widal reaction. (b) *Miliary Tuberculosis.*—The pulmonary symptoms and signs, the signs of an associated pleurisy or pericarditis, and choroidal tubercles when present suffice to establish the true nature of the affection. (c) *Septic Conditions.*—Local necrotic processes, multiple foci of inflammation, irregular chills, fever and sweating, embolic phenomena are diagnostic. 3. Intoxications.—Uræmic coma may be mistaken for poisoning by alcohol or opium. The anamnesis is important. In all cases the urine must be drawn and examined. The circumstances in which the patient is found and the odor of the breath may be suggestive. (a) *Alcohol.*—The temperature is subnormal, the pupils usually dilated, the coma often incomplete; if it alternates with delirium the latter is of peculiar type and attended with tremor. (b) *Opium.*—Contracted pupils, slow pulse and respiration, profound stupor from which the patient cannot be roused favor the diagnosis of opium poisoning rather than uræmia.

Prognosis.—As uræmia is a secondary toxæmia the prognosis depends upon that of the primary disease. Latent uræmia dependent upon anuria may disappear when the flow of urine is re-established. The uræmia of acute nephritis disappears with recovery from the renal condition; that of the chronic forms may be transient and recurrent. Sudden uræmic coma is common in chronic interstitial nephritis, and may be the first indication of renal disease. It is frequently fatal.

V. THE NEPHRITIDES.

(a) Acute Nephritis.

Acute Parenchymatous Nephritis; Acute Diffuse Glomerulonephritis.

Definition.—Acute diffuse inflammation of the kidneys, caused by the action of cold, poisons, or the toxins of the infectious diseases, and characterized by scanty urine containing albumin, blood-corpuscles, and tube-casts, a tendency to dropsy, and evidences of toxæmia.

Etiology.—Exposure to cold and wet is very often followed in the course of a day or two by the evidences of acute nephritis. Trench diggers and other laborers in low wet places are especially liable. It is common after the exposure incident to a debauch. Certain drugs, as cantharides, internally administered or externally applied, turpentine, balsam of Peru,

potassium chlorate, naphthol, and certain acids, as sulphuric acid, salicylic acid, and phenol, in excessive doses are sometimes followed by this form of nephritis. The nephritis which frequently follows scarlet fever is typical. Less common and usually less intense is the acute nephritis associated with pneumonia, enteric fever, influenza, and diphtheria. The acute nephritis of yellow fever and cholera is of severe type. Nephritis may occur in association with variola, varicella, meningitis, syphilis, septic conditions, purpura, and angina tonsillaris. The most important infecting organism is the streptococcus, especially *S. Viridans*. Localization in the glomeruli is the primary renal lesion. It is probable, however, that a streptococcus toxæmia without localization may in some cases cause nephritis. The acute nephritis of pregnancy is usually an exacerbation of a preëxisting chronic nephropathy; otherwise the condition is a nephrosis —that is a toxic tubular degeneration without glomerular involvement. The remarkable form which occurs after extensive burns and other cutaneous lesions probably belongs to this group.

Symptoms.—The general symptoms after exposure to cold and wet usually develop suddenly; after poisoning and the infections, gradually. The onset in children may be attended with convulsions; in adults, by a chill or chilliness. Much more common are such initial symptoms as pain in the back, nausea, vomiting, and headache. Pallor, puffiness about the eyes, and œdema of the ankles are very often the first symptoms to call attention to the kidneys. Fever is not constant. It is more common in children than adults. The temperature may reach 102°–103° F. (38.9°–39.5° C.). Its range does not conform to type.

The urinary changes are characteristic. The quantity is at first greatly diminished. Anuria may occur. Usually a few ounces—100 to 200 c.c.—are secreted in twenty-four hours. The specific gravity is high—1.020 to 1.030. Later, when the secretion is re-established, the specific gravity falls to normal or below it. The percentage of urea is high, but the total quantity is greatly reduced. Owing to the excess of solids the urine is not transparent. It varies in color from a mere smokiness to the dense, opaque brown of porter. These changes are due to the presence of blood, but the urine is never bright red. Upon standing an abundant, dark, coarse sediment is precipitated, which consists of red blood-corpuscles, epithelium from the urinary tract, uric acid and other crystals, and hyaline, granular, blood, and epithelial tube casts. Albumin is abundant and upon testing precipitates in coarse, curdy flakes. Upon the application of heat the urine may solidify in the test-tube. The foregoing urinary changes are of highest grade in the beginning of the attack. They are to some extent a measure of the severity of the disease and they gradually lessen as improvement occurs in favorable cases.

Dropsy, though exceptionally absent, is a frequent and important symptom. It varies from mere puffiness about the eyelids to a general anasarca with effusion into the serous sacs. It is a peculiarity of the dropsy of acute nephritis that it is irregular in its distribution and does not always gravitate according to the posture of the patient. The degree of œdema is greater after colds, in pregnancy, and after scarlatina than after the other

infections. In the nephritis of diphtheria there may be little or none. There are cases of post-scarlatinal nephritis in which effusion into the serous cavities occurs with scanty subcutaneous œdema. Pulmonary œdema and œdema of the glottis may occur. Anæmia is an early and marked condition. Epistaxis is common and symptomatic purpura not infrequent. The pulse tension may be increased and the aortic second sound accentuated. Acute dilatation of the heart may occur. Albuminuric retinitis is comparatively infrequent, though retinal hemorrhages are occasionally encountered.

Uræmic symptoms, among which we include the preliminary anorexia, dulness, and headache, and the initial nausea and vomiting, are almost constant. When to these minor symptoms caused by the retention of excrementitious substances are added convulsions and coma, the condition of acute uræmia is fully established. This may occur at any period in the course of the attack.

Diagnosis.—DIRECT.—The general symptoms are variable and by no means characteristic. Pallor, with slight puffiness of the ankles or eyelids, may be present in the absence of subjective sensations of impaired health; or the symptoms of the causal affection may mask those of the nephritis. This is apt to be the case in pregnancy. It is therefore imperative that the urine be examined at intervals of two or three weeks as a matter of routine during gestation. The clinical picture of acute nephritis in the acute cases following cold, or occurring after scarlatina, is such as to justify a positive diagnosis. In the insidiously developing cases the conditions may be less obvious. The urinary findings as given above are of diagnostic significance. The presence of blood-corpuscles with blood and epithelial casts is characteristic.

DIFFERENTIAL.—1. *Simple febrile albuminuria* cannot in all cases be distinctly differentiated from an infectious nephritis. In favor of the former is the absence of special symptoms, pallor, slight œdema, or uræmic phenomena, and certain characters of the urine, namely, albuminuria of lighter degree and transitory duration, lower specific gravity and larger quantity of the urine, and the absence of blood-corpuscles and blood and epithelial casts.

2. *Intercurrent acute nephritis* in the course of chronic nephritis. This condition is by no means infrequent. The acute attack has the clinical phenomena of the primary affection and is often regarded as primary. Attention to the anamnesis, which shows antecedent poor health, characterized by weakness and lassitude, headache, gastric derangements, pallor, and slight or transitory œdema, and usually the absence of any recent definite causal factor, may explain the occurrence of acute dropsy with toxic phenomena. The urinary changes are less sharply defined than in the primary cases, and a tense pulse with cardiac hypertrophy and accentuated aortic second sound, and particularly albuminuric retinitis, render the diagnosis of coexistent chronic nephritis certain.

Chronic Passive Congestion of the Kidneys.—This form of kidney disease might be mistaken for early nephritis if the urinary findings were made the diagnostic criteria, but the signs of myocardial insufficiency,

dyspnœa, œdema, hepatic enlargement, cardiac dilatation, and arrhythmia are very suggestive. If the symptoms clear up under rest in bed and digitalis, the absence of nephritis may be assured. The tests of renal functions may be necessary.

Prognosis.—The outlook depends more upon the course than upon the immediate condition of the patient. It is more unfavorable in post-scarlatinal and puerperal nephritis than in other forms. Acute nephritis following cold is less dangerous. That which follows the various infections other than scarlet fever is usually of milder type. Complete and rapid recovery may follow the intense forms associated with yellow fever and cholera. The death-rate in infancy is not lower than 33 per cent. The prognosis as to entire recovery is uncertain. Acute nephritis is very often the point of departure for the chronic form. Even with apparent recovery there remains an especial liability to attacks later in life. At the onset neither the dropsy, the amount of urine, nor the proportion of albumin which it contains justifies a positive prognosis. Urgent uræmic symptoms are always alarming. In the gravest acute nephritis dropsy may be absent. Complete anuria lasting for a day or two may occur in cases terminating favorably, and dense albuminuria often gradually disappears. Low arterial tension, intense anæmia, persistence of dropsy, effusion into the serous sacs, continuing albuminuria of high grade, and chronic uræmic symptoms are of unfavorable prognostic import. Cases thus characterized are liable to an acute fatal exacerbation, or escaping that, to a chronic course. The absence of these conditions is favorable. Recovery may be practically complete in four or six weeks. In other cases a favorable termination may occur at the end of several months.

(b) Chronic Nephritis.

Anatomically two principal forms are encountered, namely, chronic parenchymatous nephritis and chronic interstitial nephritis, and these respectively manifest themselves by a more or less well-defined symptom-complex.

1. CHRONIC PARENCHYMATOUS NEPHRITIS.

Chronic Diffuse Glomerulonephritis; Chronic Desquamative Nephritis; Large White Kidney.

Definition.—Chronic diffuse inflammation of the kidneys, occurring as a sequel of acute nephritis or developing insidiously, with marked changes in the parenchyma and lesions of the interstitial connective tissue, and characterized by albuminous urine of high specific gravity containing tube-casts, œdema and secondary anæmia.

Etiology.—This form of nephritis frequently follows the acute variety and is due to the same causes. In many cases it insidiously develops after an acute attack in the course of a chronic infection. It is common in persons who have suffered from repeated attacks of malarial fever, and in chronic alcoholism. It is met with at all ages, but is especially common in early adult life, and in children as a sequel of scarlatinal nephritis. In adult life it is more frequent in males than females.

The kidneys may show the changes which constitute the large white kidney, the small white or pale granular kidney, or the kidney of chronic hemorrhagic nephritis. It has been definitely established that the pale granular kidney is a condition consecutive to the large white kidney. The renal epithelium shows degenerative changes. The tubules contain red and white blood corpuscles. The glomeruli are enlarged and show nuclear changes in the endothelial and connective tissue cells. Œdema of the interstitial connective tissue and areas of round-celled infiltration may be seen.

Symptoms.—When consecutive to the acute form, chronic parenchymatous nephritis presents similar, though less urgent, symptoms. A majority of the patients, after a period of failing health with ill-defined symptoms, become pallid and puffy about the eyelids and ankles, or the albumin and casts are discovered upon routine examination of the urine.

The urine, especially in the earlier course of the disease, is diminished in quantity, averaging in the adult 750 to 1000 c.c. in the course of twenty-four hours. With increasing dropsy the output usually diminishes, to again increase as the dropsical effusion undergoes resorption. It is dirty yellow in color and turbid from the presence of urates. Upon standing it precipitates a sediment, which reveals upon examination leucocytes, often red blood-corpuscles, epithelium from the urinary passages, and many tube-casts, hyaline, granular, fatty, and epithelial. Albumin is usually abundant, showing a relative decrease during repose and an increase after exercise. It varies in amount from .5 to 2 per cent. The specific gravity is above normal, varying from 1.025 to 1.035. As the case progresses, the average daily quantity of urine augments, while the average daily amount of albumin excreted declines, and the specific gravity falls. The daily amount of urea fluctuates, but the average is below the normal. The presence of blood in considerable quantities is suggestive of chronic hemorrhagic nephritis.

The tendency to dropsy is characteristic of this form of nephritis. The eyelids and ankles early in the disease, the subcutaneous tissues generally later, and toward the close the serous sacs become the seat of dropsical effusions. The loose tissues of the genitalia in both sexes, and dependent parts in general, become in advanced cases highly distended and œdematous. Uræmic symptoms, especially headache and drowsiness, are common. Gastro-intestinal symptoms, anorexia, nausea, vomiting, and diarrhœa belong to this group. Convulsions may occur as the end approaches. Albuminuric retinitis is not common early in the disease. Hypertrophy of the heart is of less frequent occurrence and less marked than in the chronic interstitial form. The longer the duration of the disease the greater the tendency to cardiac enlargement. The blood-pressure is usually increased, the aortic second sound accentuated, and some degree of arterio-sclerosis gradually develops. In chronic parenchymatous nephritis there is a peculiar tendency to bronchitis, pneumonia, pleurisy, and pericarditis.

A protracted course, with the symptoms and urinary changes of chronic parenchymatous nephritis passing by degrees into urine suggestive of the chronic interstitial form, would justify the conclusion that the large white

kidneys were undergoing changes into the small white kidney as a later stage.

Diagnosis.—Direct.—The recognition of this form of nephritis depends upon the association of the above-described urinary changes, the tendency to dropsy, and mild uræmic symptoms, as headache, drowsiness, loss of appetite, and nausea. In mild cases the diagnosis must rest upon the condition of the urine. The facies, which exhibits marked pallor, a dull, pasty complexion, and puffy eyelids, is suggestive.

Prognosis.—The prognosis is grave, both as to life and as to recovery. A limited proportion of cases under very strict and judicious management, especially among children, recover in the course of a year or two. The greater number have already, when the diagnosis is made, entered upon a life of chronic invalidism. The scene closes with increasing and unmanageable dropsy, acute uræmic symptoms, or intercurrent acute inflammation, usually of the pleuræ or pericardium.

2. CHRONIC INTERSTITIAL NEPHRITIS

Contracted or Granular Kidney; Sclerosis of the Kidney; Gouty Kidney.

Definition.—Chronic diffuse inflammation of the kidneys, associated with overgrowth of intertubular connective tissue, and characterized by insidious development, increased urine of low specific gravity, albuminuria, which is usually slight and often intermittent, arteriosclerosis, and little tendency to dropsy.

Anatomically, there is marked increase in the connective tissue, with degeneration of the parenchymatous structures. The process is essentially atrophic, the atrophy probably beginning in the glomerules and tubules, and being followed by increase in the connective tissue. Three forms are described: (a) The pale granular kidney, to which reference has already been made—*secondary atrophic kidney;* (b) the contracted kidney, occurring as an independent affection—*primary atrophic kidney;* and (c) sclerosis of the kidneys, developing in connection with cardiovascular disease—*arteriosclerotic atrophy of the kidney.*

Etiology.—The pale granular kidney probably constitutes a later stage of the large white kidney, in which atrophic changes in the secreting structures have been followed by connective-tissue overgrowth. The primary contracted kidney is the result of a gradual degenerative process. Many of the cases occur in the absence of discoverable cause and may be due to premature senile involution. Focal infection is probably a common cause. Early arteriosclerosis, syphilis, and gout are causal factors. Chronic intoxications, especially alcohol and lead, play an important part in the etiology of sclerosis of the kidney. Habitual excesses at table, especially the eating of large quantities of meat, are particularly injurious. The arteriosclerotic form is associated with cardiac hypertrophy and sclerosis of the arterial walls. It is common in active, energetic men, who work hard and eat and drink too much. Habitual anxiety and worry, and inability to endure the stress of life favor the development of the vascular changes of which contracted kidney is the outcome. This form of renal disease is much

more common after forty than in early life, and in men than in women. In this country arteriosclerosis develops at an earlier age among the Slav immigrants than in persons of other nationalities. Hereditary influences are important. In fact the form of so-called chronic interstitial nephritis corresponding to the *primary atrophic kidney* occurs with such frequency in successive generations as to suggest a familial causative factor. I have knowledge of several families in which almost every member in three and in some cases probably four generations has developed this disease in early middle life.

Symptoms.—The changes in the kidneys are insidious and frequently reach an advanced stage before they cause symptoms that attract attention. Acute uræmic symptoms frequently constitute the first marked manifestations of the disease. The earlier symptoms, headache, frequent micturition, and digestive disorders are often disregarded by the patient. More commonly the general health becomes gradually impaired. The patient is weak and breathless upon exertion. He rises frequently to pass urine, suffers from habitual headache, and complains of nausea and occasional vomiting.

The composition of the urine varies to some extent according to the variety of the renal atrophy. In the secondary form the quantity of the urine is less and the amount of albumin greater than in the primary form. The specific gravity is more nearly normal, that is, higher. There are various kinds of casts and a few red blood-corpuscles in the sediment. The tendency to dropsy is more marked. In the primary form the quantity of urine is much increased above normal and frequently reaches as much as four litres. This polyuria is the cause of an abnormal thirst. The color is pale yellow, the transparency clear, and the specific gravity low—1.005 to 1.012. A scanty sediment is deposited, in which are found a few hyaline and granular tube-casts, granular epithelial cells, leucocytes, and rarely red blood-corpuscles. The amount of albumin is small, especially after repose. It increases during attacks of intercurrent disease or with cardiac weakness, when the quantity of urine is diminished. It is characteristic of this form of nephritis that there are often albumin-free periods, especially in the early part of the day. The casts in some instances disappear even while the urine remains albuminous. The urinary solids are decreased, especially urea. Temporarily the urea may reach normal, and uric acid, phosphoric acid, the chlorides, and ammonia may approach normal. In the arteriosclerotic form polyuria is less common, the color of the urine is normal, the albumin is more abundant and more constant, and there are hyaline and granular casts, which may at times disappear.

Dropsy, so long as the power of the hypertrophied heart and the polyuria are maintained, is absent or scanty. Pretibial œdema may be noted, or slight puffiness of the ankles. The heart is hypertrophied, the left ventricle being first affected. The apex is displaced to the left and downward. The impulse is forcible and sometimes heaving. The aortic second sound is accentuated. There may be reduplication of the first sound, or an apex systolic murmur transmitted to the axilla. Toward the close the hypertrophy fails and the signs of dilatation are pronounced, together

with lessened urine, increased albumin, and mounting dropsy. The pulse is hard and tense. The superficial arterial walls are thickened and incompressible. The radials can be rolled with the finger like a whip-cord upon the underlying bone. The temporals are prominent and tortuous. There is early and persistent increase of blood-pressure. Epistaxis is common and may be troublesome. Hemorrhages into the skin occur. Headache is a very common symptom. The symptoms in advanced cases are mostly due to the cardiovascular conditions, or to uræmia. To the former group are to be referred sudden œdema of the glottis or lungs, pleural effusion, and some cases of cardiac dyspnœa. This symptom may resemble asthma and is often troublesome at night. Cerebral hemorrhage is not uncommon. Fully 40 per cent. of the cases of apoplexy occur in persons suffering from contracted kidneys. To uræmia, either in its chronic or acute forms, must be referred certain of the cases of nocturnal dyspnœa—so-called renal asthma, Cheyne-Stokes respiration, nausea, vomiting, which is often uncontrollable, and diarrhœa. The complexion is usually pallid and muddy. Sweating is uncommon. The urea "frost" may be deposited after free perspiration. Pruritus and eczema are common. Muscular cramps occur, especially on waking in the morning. Albuminuric retinitis occurs more frequently than in any other form of nephritis. Visual troubles, in a large proportion of the cases, lead to the discovery of the actual condition. Sudden blindness without ophthalmoscopic findings—uræmic amaurosis—is sometimes observed. Hemorrhages beneath the conjunctivæ or into the eyelids occur. Tinnitus aurium or cerebri, vertigo, and nervous deafness are encountered.

Persons subject to chronic interstitial nephritis are peculiarly liable to severe intercurrent diseases. Bronchitis and pneumonia are common. Inflammatory affections of the serous membranes, as acute pleurisy and pericarditis, occur.

Diagnosis.—The early stages of chronic interstitial nephritis present no characteristic clinical phenomena. The anatomical condition may be advanced in cases unattended by evidences of ill health prior to the occurrence of the acute disease which has been the cause of death. The association of cardiac hypertrophy, sclerotic arteries, high pulse tension, accentuated aortic second sound, with copious urine of low specific gravity containing an inconstant trace of albumin and a few hyaline and granular casts, justifies a positive diagnosis. The urine should be repeatedly examined, specimens being taken at night and in the morning. The condition is often discovered accidentally in examination for life insurance, and sometimes overlooked under the same circumstances.

The diagnosis of small granular kidney cannot be positively made from the symptoms and urinary composition. It is rendered probable by a previous history of acute or chronic parenchymatous nephritis. Nor can the arteriosclerotic form be distinguished from the other varieties with certainty. The diagnosis becomes probable when the patient is past forty and has marked cardiac hypertrophy, hardened arteries, increased pulse tension, and uræmic symptoms, and particularly when the progress of the

case is comparatively rapid. Functional tests are of some aid in determining the progress of the process. The phenolsulphonaphthalein permeability is moderately diminished.

Prognosis.—The outlook as regards recovery is hopeless. The disease is incurable. As regards prolongation of life and a fair degree of health, the prognosis is not altogether without encouragement. Many of the cases, under careful management and with a self-denying and regular manner of living, make slow progress and continue for years without passing into invalidism. The symptoms of chronic uræmia are danger signals; those of acute uræmia heralds of catastrophe. The signs of cardiac failure are usually the beginning of the end.

VI. TUBERCULOSIS OF THE KIDNEY.

Definition.—There are two forms: (a) miliary tuberculosis of the kidney occurring in connection with general miliary tuberculosis and (b) local renal tuberculosis arising as a secondary tuberculous process in focal tuberculosis in some other organ or structure, as a lymph gland, bone caries, the lungs, and so on. The first of these is of little clinical interest. The second is of great practical importance from the standpoint of diagnosis and surgical treatment.

Etiology.—Predisposing Influences.—Local renal infection is liable to occur in any case of tuberculosis near or remote. Malformation or displacement of the kidney, traumatism, cystitis, calculus or local inflammation, the lesions of the various forms of substantive renal diseases constitute predisposing influences of importance. It may occur at any age but is more common in early middle life and in females than in males.

The Exciting Cause.—Infection by the bacillus tuberculosis. The view at one time held that the infection takes place by continuity of structure lacks the support of present knowledge. Such ascending or contiguous infection is very rare. Hematogenous infection is the rule. Cases in which the kidneys and the genital organs have been affected at the same time have shown an absence of signs of the extension of the tuberculous process from one set of organs to the other. In cases in which the diagnosis is made early about 90 per cent. show involvement of one kidney, usually the right; later both kidneys are often affected. A descending infection of the ureter and bladder may occur.

Symptoms.—Urinary derangements are among the earliest symptoms. These consist of frequent micturition, burning and tenesmic pain towards the end of the flow; nycturia and polyuria. The urine is acid, albuminous and contains white and red blood corpuscles. Blocking of the ureter on the affected side may occur, with a temporary passage of normal urine. Tubercle bacilli are frequently present. Smegma bacilli and other acid-fast bacilli are to be differentiated. Hyaline and granular casts are often present. Hematuria with ureteral blood casts are met with. Pain and palpable swelling of kidney are inconstant. Röntgenographic examination may reveal enlargement of the kidney. Constitutional symptoms indicative of a general impairment of health are present. Their importance from a diag-

nostic standpoint arises from the necessity of ascertaining their cause. The course of neglected cases is chronic and progressive.

In uncomplicated cases leukopenia is commonly present.

Diagnosis.—DIRECT.—The early recognition of the renal disease depends upon the habitual routine examination of the urine in every patient. The recognition of the above symptom-complex calls for the search for tubercle bacilli in the urine by animal inoculation if necessary. Cystoscopic examination is important to determine whether one or both kidneys are involved.

DIFFERENTIAL.—Renal tuberculosis must be differentiated from pyelitis, pyelonephritis, nephrolithiasis, from hypernephroma and when there is hematuria from hemorrhagic nephritis, carcinoma of the kidney, scurvy, and essential hematuria. A careful differentiation must be made between renal tuberculosis and ordinary nephritis, which is a very common associated disease in tuberculosis.

Prognosis.—The course of the disease in untreated cases is chronic and progressive. The early extirpation of a tuberculous kidney frequently saves suffering and greatly prolongs life.

VII. SYPHILIS OF THE KIDNEY.

There are two forms: (a) diffuse nephritis and (b) gumma of the kidney. The symptoms are not characteristic and the diagnosis at best only a probable one.

VIII. PYELITIS.

Definition.—Inflammation of the pelvis of the kidney, due to direct bacterial infection by way of the blood or the ureters and lymphatics.

When the inflammation extends to the substance of the kidney the condition is designated pyelonephritis; when the entire organ is involved, pyonephrosis or renal abscess; the form due to tuberculosis is known as nephrophthisis.

Etiology.—Under ordinary conditions the kidneys are capable of eliminating, without damage to themselves, the pathogenic organisms reaching them by way of the blood stream or ureters and lymph channels in constitutional or local infection. When, however, their resistance to pathogenic influences is diminished by such general causes as prolonged malnutrition, anæmia, cold, or over-exertion, or by local conditions, as congestion, nephritis, pressure upon the kidney or ureter, twisting of the ureter in displacement or operation, infection occurs. Whether this takes place from the side of the blood current or from the urinary tract, the pelvis of the kidney is first affected—*pyelitis*. The colon bacillus, Bacillus proteus, streptococcus, and Staphylococcus albus have been found in pure cultures. The tubercle bacillus is the cause of a special form of pyelitis. That form which occurs in gonorrhœa is caused not by the gonococcus but by associated pyogenic organisms.

Morbid Anatomy.—Pyelitis may be catarrhal or suppurative. The tuberculous form begins locally, the kidney gradually becoming infil-

trated with tubercle which undergoes caseation and softening, with ultimate transformation into cretaceous masses from the resorption of fluid elements and the deposition of lime salts. It is associated with tuberculosis of the ureters, bladder, and prostate and testicles, or the ovaries or Fallopian tubes—*urogenital tuberculosis*. Pyelitis due to local causes usually affects one kidney; that caused by general conditions may involve one or both; the form consecutive to cystitis, following enlarged prostate, stricture, catheter infection, and surgical operation, is bilateral and extends to the kidney substance. The acute suppurative pyelonephritis which follows operations is known as *surgical kidney*.

Symptoms.—The pyelitis which accompanies the acute infections usually causes no symptoms by which it can be recognized during life. There may be pain in the back and deep tenderness over the affected kidney. The urine is albuminous, turbid, sometimes acid, sometimes alkaline, and contains a few pus-cells, transitional epithelial cells, and red blood-corpuscles, rarely tube-casts. Recurrent attacks occur in which, after an interval during which the patient has had clear urine and no special symptoms, the urine suddenly becomes turbid and smoky, and contains albumin and pus-cells, the change being accompanied by pain in the lumbar region, chills, fever, and profuse sweating.

In chronic pyelitis the pus in the urine varies in amount and may at times wholly disappear—a phenomenon due to the blocking of the ureter when one kidney only is affected, and associated in some cases with the signs of a tumor in the renal region. In acute pyelonephritis shreds of renal tissue are sometimes present, together with tube-casts which may be composed of pus-cells or bacteria. The urine is usually increased in amount and contains albumin in proportion to the pus and blood present. Its reaction varies, being usually alkaline, but sometimes acid, according to the infecting bacterium. It is commonly acid in the clear intervals when the pyuria is intermittent, and alkaline when there is an associated cystitis.

Paroxysmal fever, intermittent in type and associated with chills and sweating, is very common. The attacks are sometimes ague-like and recur with a periodicity so regular that they closely simulate malaria. After a time the chills cease and the fever assumes the hectic type. Chronic pyelitis is usually accompanied by emaciation, anæmia, and progressive impairment of health. Sepsis with secondary abscess formation may develop—septicopyæmia. The symptoms in some cases suggest enteric fever, but the diagnostic clinical and laboratory criteria of that disease are wholly lacking. There is a considerable group of cases familiar to practitioners in which, with intermittent or persistent pyuria, fairly good general health is maintained. A knowledge of this fact is important in connection with surgical considerations. Dryness of the mouth, vomiting, profound asthenia, and drowsiness passing into coma, with dyspnœa—a condition suggestive of diabetic coma—sometimes constitute a terminal symptom-complex. At one time attributed to intoxication by ammoniacal products of decomposing urine, this condition has been regarded as an ammoniæmia. It is probably due to intoxication products of decomposing urine or pus, or specific bacterial toxins. It differs from uræmia in the

absence of convulsions and retinitis. Paraplegia, variously ascribed to myelitis, peripheral neuritis, or reflex causes, is not uncommon.

The local swelling in the renal region varies in size from time to time. It may attain large size and give rise to signs of fluctuation in pyonephrosis —*abscess of the kidney.*

Diagnosis.—Direct.—The constant or intermittent presence of pus and blood in the urine, the occurrence of renal tissue, the absence of tube-casts, a tumor in the renal region inconstant in size or showing deep fluctuation in the absence of œdema, one-sided lumbar pain and tenderness, and chills, fever, sweating, wasting, and anæmia justify a positive diagnosis. In the absence of several of these clinical phenomena a provisional diagnosis may be made.

Differential.—Tuberculous pyelonephritis may be diagnosticated when, with the above symptoms, tubercle bacilli are present in the purulent urine. In doubtful cases laboratory methods must be employed, especially the inoculation of guinea-pigs. Evidences of tuberculous disease in the urinary passages or genital organs are of diagnostic importance. The discrimination between pyelitis and pyelonephritis cannot always be made with precision. The presence of minute bits of renal tissue in the urine or a tender tumor in the region of the kidney would point to the latter condition. A deep fluctuating tumor points to renal abscess. Abscess within the capsule of the kidney is to be distinguished from perirenal abscess by the more circumscribed outline of the tumor, the absence of œdema, and the anamnesis, but the differential diagnosis is sometimes impracticable.

Cystitis and pyelitis are frequently associated. The polyuria, intermitting pyuria when present, the pain, tenderness, and tumor mass in one lumbar region, and the absence of frequent micturition and vesical tenesmus are in favor of the latter affection. The anamnesis is important. The cystoscope and catheterization of the ureters may be employed in doubtful cases.

Prognosis.—The cases associated with the acute febrile infections usually recover with the convalescence from the primary disease. The tuberculous form, when the kidney only is infected, may terminate in recovery, with cretaceous masses replacing more or less renal tissue. In abscess the outlook is unfavorable. Amyloid disease, fatal sepsis, or peritonitis from perforation may occur. The diagnosis assumes importance in view of the possibility of relief by surgery.

IX. PERINEPHRIC ABSCESS.

Paranephritis; Perirenal Abscess.

Definition. — Suppurative inflammation of the connective tissue surrounding the kidney.

Etiology.—Perinephric abscess may follow blows and injuries, the acute febrile infections, especially in children, inflammation of the kidney or ureter, perforation of the appendix or bowel, or result from a perforating empyema or spinal caries.

Morbid Anatomy. — The pus cavity is usually extensive, and the adjacent tissues œdematous. The accumulation is usually posterior, but may be anterior, to the kidney. It shows a strong tendency to burrow, and may perforate into the pleura, the bowel, the peritoneum, the bladder, or vagina, or follow the direction of gravitating spinal pus along the sheath of the psoas muscle or the iliac fascia, or finally the abscess if left to itself may burst externally.

Symptoms.—Pain in the region of the kidney, aggravated by pressure, or referred to the hip-joint, or inside of the thigh, and associated with retraction of the testicle, a limping gait, flexed thigh, stooping posture, and rigid spine, deep induration and œdema, and a tumor mass upon palpation between the last rib and the crest of the ilium, normal urine unless the primary pus depot is within the capsule of the kidney, and the constitutional evidences of pus make up the clinical picture.

Diagnosis.—The DIRECT DIAGNOSIS is justified by the above association of symptoms. Pus-free urine, and œdema overlying the tumor, and deep fluctuation are significant. It is not always possible to determine whether the infection comes from the kidney or some source outside of it. Here the history is important.

DIFFERENTIAL. — The pain and attitude may closely simulate hip-joint disease; but the essential symptoms are wanting, and the tumor and œdema in the region of the kidney are conclusive. When the abscess points in the inguinal region spinal caries must be excluded.

X. NEPHROLITHIASIS.

Renal Calculus; Renal Infarct.

Definition.—A condition characterized by the presence in the substance or pelvis of the kidney, or in the ureter, of concretions formed by the deposition of certain of the constituents of the urine normally held in solution.

RENAL INFARCT.—The deposition of urinary salts in the substance of the kidneys occurs under the following circumstances: In the new-born in the shape of uric acid crystalline masses in the tubules, and especially at the apices of the pyramids—*uric acid infarcts;* in chronic gout, sodium and ammonium urate in whitish linear deposits, chiefly in the pyramids—*sodium urate infarcts;* and dense white linear deposits of calcium phosphate or carbonate in the pyramids, chiefly in aged persons—*calcareous infarcts.*

RENAL CALCULUS.—The concretions which form in the pelvis and calices constitute, according to their size, sand, gravel, or stone. Renal sand consists of gritty particles of a size not too large to traverse the urinary passages without arrest. These minute calculi are frequently voided in the urine in considerable quantities at intervals for years, without causing symptoms. They form a characteristic coarse urinary sediment. The term gravel is applied to larger concretions, ranging in size from a canary seed to a pea, usually multiple, sometimes single, round and smooth, or irregular and rough, which form in the pelvis and calices, and passing into the ureter cause renal colic. Kidney stones, dendritic or coral calculi, are larger concretions, which attain dimensions in the pelvis which prevent

their entering the ureter. Gradually increasing in size, they often form remarkable branching casts or moulds of the pelvis and calices. *Ureteral Calculi.*—The orifice of the ureter may be blocked by a large calculus, formed in the pelvis of the kidney, or a calculus may be arrested at any point in its course from the kidney to the bladder.

Chemical Composition of Renal Calculi.—These concretions do not represent mere precipitations of crystallizable or other solid constituents of the urine. They are composed in the first place of mixtures of various substances of this kind, arranged irregularly or in concentric layers, and in the second place they contain various proteid substances present as the result of inflammatory irritation of the tissues with which they are in contact, and finally bacteria are frequently found in the somewhat irregularly differentiated central nucleus. The principal varieties consist of: *Uric Acid and Urates.*—This is the common form and constitutes renal sand, small single calculi, and the large branching kidney stones. They are of a yellowish or brownish-red color, smooth or slightly irregular surface, and dense consistence. The large stones are composed of concentric strata and are very hard. They consist of uric acid and urates in varying proportions. In urinary sand the crystalline particles may consist of uric acid alone; in children calculi composed of urates occur. *Calcium Oxalate.*—Mulberry calculi are of a brownish or black color, rough and mammillated or pointed surface, and very hard. They are composed of oxalate of lime and uric acid, the former predominating. *Calcium Phosphate and Ammoniomagnesium Phosphate.*—Phosphatic calculi are composed of these salts, together with small quantities of calcium carbonate. They are of a whitish or pale fawn color, crystalline or chalk-like surface, and light consistence. They are sometimes friable. They are common, and the substances of which they are composed are deposited as an outer layer upon uric acid or oxalate calculi. *Cystin and Xanthin.*—Calculi chiefly composed of these substances are occasionally met with. Urate, phosphate and oxalate calculi are very common; the other varieties are rare.

Analysis of Urinary Calculi.—Organic calculi are completely incinerated on platinum; inorganic calculi are not. *Murexid Test.*—Uric acid or urates.—Fragments of the mass are placed upon a porcelain dish and a drop or two of nitric acid added. Heat is then applied at first briskly but later gently to complete dryness over the water bath. At first the color is yellowish but with thorough drying there is a reddish tinge. The adding of a drop of ammonia changes the color to a purple red. Xanthin.—The color is yellow. Cystin.—The powdered mass burns with a faintly blue flame and odors suggestive of fatty substances. Calcium Carbonate.—Effervescence with hydrochloric acid. Calcium Oxalate.—No effervescence with HCl until after incineration. Early Phosphates.—No effervescence with HCl after incineration.

Immediate Effects upon the Kidney.—In many cases the kidney manifests a remarkable tolerance for the slowly forming calculus. Single or multiple stones may be found post mortem without lesions of the kidney or a history of renal symptoms. Urinary sand or small round uric acid calculi are often passed at intervals by persons otherwise in good health.

Large dendritic calculi cause induration and atrophy of the kidney substance. When infection takes place, calculous pyelitis and pyonephrosis result.

Etiology.—The subjects of uric acid and calcium oxalate crystals are usually adults of good constitution, active, and given to the pleasures of the table. Many of them are gouty. The urine is highly acid and contains uric acid in excess. Phosphatic calculi are met with in anæmic persons in poor health, often women with alkaline, sedimentary urine. In general, renal calculus is much more common in men than in women; in infancy and late adult life than in the middle periods. Dyspepsia, migraine, and a sedentary life are predisposing influences.

Symptoms.—Nephrolithiasis may be latent. Persons pass renal sand occasionally without local or general derangement of health. Sometimes a large dendritic calculus is unattended by symptoms. As a rule two sets of definite symptoms occur.

1. Symptoms of Stone in the Kidney.—These are: (a) *Pain,* which may be dull and continuous, or paroxysmal. It is usually referred to the lumbar region of the affected side, and sometimes radiates to the scrotum or glans penis. It may extend to the opposite side of the back. It may be aggravated by a misstep or sudden jarring of the body. Nephralgia is common in movable kidney, and occurs in the absence of renal calculus. (b) *Hæmaturia.*—Microscopic blood in the urine is very common. The amount of blood may cause smokiness, or, exceptionally, give a bright red tinge to the urine. Hæmaturia may be continuous, or it may recur at intervals, coming on spontaneously or after exertion, and ceasing upon rest. Bleeding is more common when the calculus is lodged in the ureter than when it remains in the pelvis of the kidney. (c) *Pyuria.*—Pus in the urine is an indication of calculous pyelitis, which may exist for a long time without causing serious symptoms, or may result in pyelonephritis or renal abscess. (d) *Septic Phenomena.*—There are cases in which paroxysms of intense pains, with chills, high fever—104° to 106° F. (40°–41.1° C.)—and sweating occur at varying but irregular intervals. The urine becomes turbid and contains blood and transitional epithelium, but remains free from pus. These attacks, often regarded as malaria, are more like hepatic fever. Their recognition depends upon the positive evidence of nephrolithiasis and negative signs as regards malaria, *i.e.,* absence of blood parasites and failure of quinine. When calculus is established, irregular chills, fever and sweating, anæmia, wasting, pyuria, and smoky urine are commonly present.

2. Symptoms of Renal Colic.—The attack begins with dull pain in the renal region, which presently extends to the flank and toward the groin. This pain is continuous with excruciating exacerbations and points of focal intensity in the glans penis and testicle, which is retracted. It is accompanied by a most urgent continuous vesical tenesmus and desire to pass water, which is without result beyond a few drops of bloody urine, the avoidance of which is attended with distressing scalding sensations. Rectal tenesmus and intense nausea and vomiting frequently add to the

distress of the patient. Restlessness, anxiety, pallor, shivering, cold sweats, feeble pulse, and other collapse symptoms are usually also present. Fever may occur, 101°–103° F. (38.3°–39.5° C.). The attack ceases abruptly, with sensations of relief as the calculus passes into the bladder or returns into the pelvis of the kidney. Its duration varies from about an hour to a day or more. In the longer attacks there are periods of remission.

During the attacks an acute hydronephrosis develops upon the affected side, which is relieved upon the escape of the calculus into the bladder with the discharge of a large quantity of urine. A large amount of clear urine may be discharged from the sound kidney during the course of the attack. Renal colic does not always terminate in complete relief. The calculus may become impacted in the ureter and cause hydronephrosis and hæmaturia; in extremely rare cases it may rupture into the peritoneal cavity or the intestine, or may form an abscess and perforate the skin. Again, anuria may occur under the following circumstances: with a normal kidney on the opposite side from functional arrest, in consequence of nervous irritation; with a previously diseased kidney from the same cause; with a single kidney. These conditions are rare.

Death occurs from uræmia, as a rule within ten or twelve days after complete anuria has set in, exceptionally not for two or three weeks.

Diagnosis.—Direct.—Other forms of paroxysmal pain may be mistaken for renal colic, but the seat of the pain, its definite extension toward the groin, with local intensification in the testicle and glans, together with hæmaturia, are characteristic. When direct evidence of the existence of a calculus, as previous attacks with the passage of one, or the voidance of one subsequent to the attack, can be secured, the diagnosis becomes certain. The recognition of a calculus in the pelvis of the kidney or in the ureter, or the grating of several calculi upon palpation, has been mentioned, but I have no personal knowledge of such signs.

Differential.—Similar paroxysms of colicky pain are attributed to accumulation of renal sand, uric acid, or oxalates in the pelvis of the kidney. Dietl's crises, the nephritic crises of tabes, and clots of blood in renal hemorrhage, such as occur in cancer, may closely simulate renal colic. Biliary colic and intestinal colic rarely give rise to uncertainty. The differential diagnosis must be reached by a careful study of the individual case. A diagnosis by exclusion may be necessary.

The diagnosis of nephrolithiasis depends upon the foregoing symptoms, the occurrence of renal colic, and the shadows cast by the Röntgen rays. This means of diagnosis is imperatively required in order to determine the presence and position of calculi, and whether they exist in one or both kidneys in connection with the considerations of surgical intervention.

The differential diagnosis between renal and vesical calculus may sometimes be in doubt. In the latter the pain is usually bilateral, more common at the neck of the bladder. The tenesmus is continuous, with frequent micturition, and the sound will detect the presence of the stone.

Prognosis.—Many cases are recurrent. There is the ultimate danger

of calculous pyelitis. The more serious accidents of renal colic and actual obstruction are rare. Many lives have been saved by surgical procedures.

XI. HYDRONEPHROSIS.

Definition.—A collection of urinary fluid in the pelvis and calices of the kidney due to obstruction of the ureter, forming a cyst by the distention and atrophy of the organ.

Etiology.—Hydronephrosis may be congenital owing to developmental defects, and may be of sufficient size to interfere with parturition. Much more commonly it is acquired. The obstruction may be in the ureter or in the bladder. In suddenly developing polyuria the normal ureter may be incapable of carrying off the excess of urine, and an acute hydronephrosis result. The ureter may be occluded by calculus, stricture following ulcer, or torsion or kinking in movable kidney. It may be blocked by neoplasms, particularly tubercle or cancer. Compression from without, by cicatricial adhesions or bands, or by abdominal or pelvic tumors, is a more common cause. Bilateral hydronephrosis may result from the implication of both ureters by any of the lesions just named. When, however, the obstruction is in the bladder, the hydronephrosis is almost always double. The common conditions are cancer, which may involve only one ureteral orifice, habitual urinary distention of the bladder in prostatic hypertrophy or stricture, and the extreme thickening and contraction of the bladder wall which accompanies these conditions.

The fluid accumulates, causing distention of the ureter above the obstruction, but especially of the pelvis of the kidney. Pyelitis may occur, but usually the kidney substance undergoes a simple, progressive atrophy, forming in extreme cases a large cyst, upon the inner surface of which traces of renal tissue may be found. As the secretion of urine diminishes mucus and serum accumulate until the cyst contains a pale yellow or straw-colored fluid, holding in solution traces of the urinary solids and frequently a little albumin. There may be turbidity from the presence of pus. The cyst thus formed may attain an enormous size and simulate ascites. Cardiac hypertrophy is frequently associated with hydronephrosis. Exceptionally complete occlusion of the ureter is followed by atrophy of the kidney without dilatation.

Symptoms.—The urinary conditions are by no means constant. In one-sided hydronephrosis with complete occlusion the urine from the vicariously acting opposite kidney may be normal in quantity and composition. When the condition is bilateral, oliguria and, ultimately, complete anuria result and death occurs with uræmic symptoms. Intermittent hydronephrosis arises when a valve-like obstruction yields to the pressure of the accumulated fluid, or the twisted or kinked ureter is restored to its natural condition. Under these circumstances there is a sudden large discharge of clear urinary fluid with the subsidence of the tumor. When the obstruction recurs the cystic tumor reappears, to vanish again when the obstruction is relieved—*flush-tank symptom.* Intermittent hydronephrosis may continue for years. It is unilateral, commonly associated

with movable kidney, and usually occurs in women. In cases in which pyelitis exists the urine may be normal when the tumor is forming and turbid from the presence of mucus, pus, or blood as the tumor subsides.

Physical Signs.—When the tumor attains sufficient size it may be recognized upon palpation or inspection. If unilateral and of moderate size, it occupies the renal region; when large it may simulate ovarian or other cysts; an enormous hydronephrosis may be mistaken for peritoneal effusion. It may also simulate solid tumors of the kidney or enlarged retroperitoneal glands. The physical signs of hydronephrosis and a large renal abscess are the same. The latter is, however, usually attended by septic phenomena. They consist of dulness in the renal region with deep fluctuation. When the tumor presents anteriorly, the ascending colon on the right side, the descending colon on the left, usually yields an oblique band of tympany.

Diagnosis.—Direct.—Hydronephrosis can only be recognized when it gives rise to a fluctuating tumor. Even then it is readily confounded with other cysts. The greatest difficulties arise when the condition involves a displaced kidney; the least, in the intermittent form. The relation of the tympanitic colon to the tumor is important. The sac may be aspirated. The fluid obtained is of low specific gravity, commonly clear, and contains urea and urinary salts, and transitional epithelium. In very old cases with extreme atrophy of the kidney substance, the fluid may contain nothing characteristic.

Differential.—*Ovarian Cysts.*—Large hydronephrosis is frequently mistaken for an ovarian tumor. The latter may be differentiated by its mobility, except in the case of hydronephrosis involving a floating kidney. Hydronephrosis has its starting-point in the renal region, whereas ovarian tumors spring from the pelvis, as can be determined by bimanual examination externally, or with the fingers of one hand in the vagina, later in the rectum, and the other hand upon the abdomen. By this mode of examination the relation of the uterine appendages and the presence or absence of a pedicle can be determined. In ovarian disease disturbances of function—for example, amenorrhœa—are not constant, just as in hydronephrosis the condition of the urine varies in different cases. *Renal Cysts.*—The differentiation is mostly impossible. In the new-born cystic kidneys and hydronephrosis may present the same signs. When the abdominal wall is congenitally defective the dilated bladder and ureters may be readily observed. Echinococcus cysts of the kidney may be suspected if daughter cysts or hooklets are found in the urine or in non-albuminous fluid obtained by exploratory puncture. But a positive diagnosis of echinococcus cyst of the kidney can only be made when it is possible to exclude such a condition perforating into the urinary passages. The hydatid thrill is by no means constant. *Mesenteric Cysts.*—These tumors vary in size from the closed fist to a cocoanut and are commonly situated in the lower right quadrant of the abdomen. They are freely movable, of oval contour, smooth surface, elastic, and fluctuating. Paroxysms of pain and vomiting are often associated with their presence. *Ascites.*—The differential diagnosis may be difficult in bilateral hydronephrosis.

Movable dulness upon change of position, the absence of tympany in the flanks, and the character of the fluid in peritoneal effusion are important.

Prognosis.—Unilateral hydronephrosis when quiescent constitutes a benign tumor, but the prognosis must in all cases be guarded. The condition acquires importance by progressive increase in size, the danger of rupture into the peritoneum or lung, pyonephrosis, and the possibility of the blocking of the other ureter, with anuria followed by uræmia. The fluid may discharge by way of the ureter and never reaccumulate. Intermittent hydronephrosis may cause little inconvenience and finally undergo spontaneous cure. When double, the condition is far more serious, and

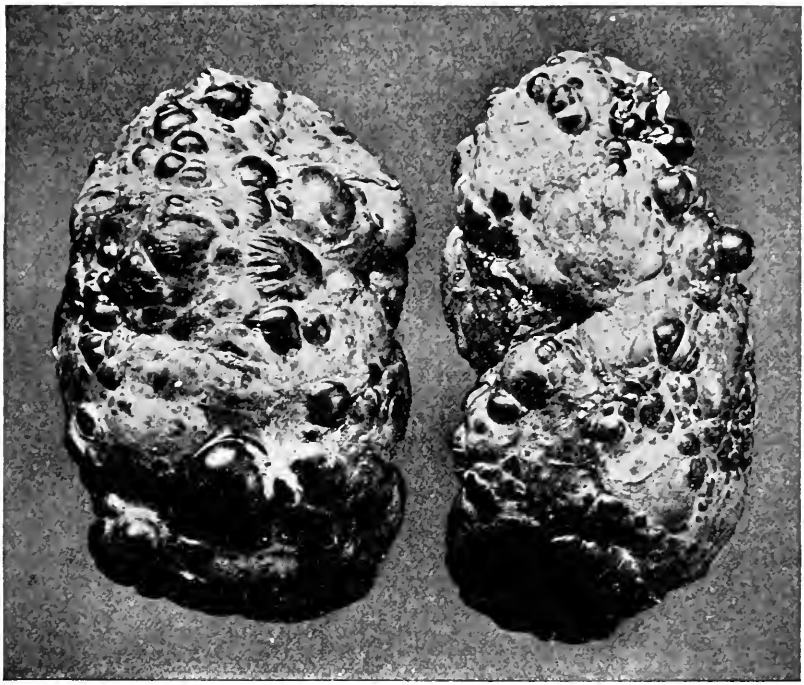


Fig. 326.—Congenital cystic kidneys.—German Hospital.

the outlook depends upon the possibility of relieving the condition which causes the obstruction—urethral stricture, prostatic enlargement, tumor-pressure. The cases due to malignant disease are without hope.

XII. CYSTS OF THE KIDNEY.

The multiple small cysts due to obstruction of uriniferous tubules in chronic nephritis, and larger solitary cysts sometimes seen post mortem in kidneys otherwise normal, do not cause symptoms or signs by which they can be recognized during life. Rare forms are combined cystic disease of the liver and kidneys, and paranephric cysts. Of greater importance clinically are: *Congenital Cystic Kidneys.*—The organs are enormously enlarged and consist of a mass of round or oblong cysts, varying in diame-

ter from 0.5 to 3 centimetres, with kidney tissue sufficient to discharge the renal function distributed in the interspaces. The fluid contained in these cysts varies in color from clear to opaque reddish or black, and in consistence from limpid to colloid. It contains albumin and other proteids, cholesterin plates, crystals of hæmatoidin and ammoniomagnesium phosphate, and fat droplets.

Etiology.—Cystic kidneys occur in the fœtus and may be the cause of dystocia. They are regarded as developmental defects. They are sometimes associated with hydrocephalus, defects of the bladder, and malformations of the extremities. The cystic condition increases with age and is encountered in young adults, the tumors often attaining great size. The condition is usually bilateral, though unilateral cases have been observed. The condition has been noted in several members of a family.

Symptoms.—The urine is abundant, of low specific gravity, and contains albumin in small amounts and hyaline and granular casts. Recurrent hæmaturia may be noted. There is pallor, a muddy skin, and not rarely diffuse cutaneous pigmentation.

Physical Signs.—The physical examination reveals the evidences of cardiovascular disease—enlargement of the heart, accentuated aortic second sound, hardening of the arteries—and the signs of double tumor in the renal region extending forward, elastic but non-fluctuating, and, when distinctly palpable through the belly walls, irregularly nodular. I have observed these signs in a unilateral case in which the diagnosis was confirmed by operation.

The symptoms, urinary conditions, and general physical signs are those of chronic nephritis; the local signs, those of renal tumors.

The **diagnosis** rests upon the association of the foregoing symptoms and signs. The recognition of the condition derives its importance from the fact that, as a rule, to which there are very few exceptions, surgical intervention is positively contraindicated.

Prognosis.—The outlook is not favorable. Death results in many cases before or directly after birth. If the patient survive infancy, death occurs before thirty from cardiovascular changes or uræmia.

XIII. TUMORS OF THE KIDNEY.

Solid tumors of the kidney are benign and malignant. Benign tumors comprise fibroma, lipoma, myxoma, angioma, gumma, and adenoma. They do not give rise to symptoms referable to the kidneys, nor do they, as a rule, attain such a size as to respond to the methods of physical examination—inspection, palpation. They are, therefore, of anatomical rather than clinical interest.

Malignant neoplasms—carcinoma, sarcoma—are primary or secondary. They are accompanied by distinct symptoms, both general and urinary, and frequently grow to a large size.

Renal carcinoma is a rare lesion and when small may not reveal its presence during life. Larger cancers cause conspicuous abdominal tumors, usually immovable upon deep respiration or palpation, occupying the

upper quadrants of the abdomen in relation with the liver or spleen, obliquely traversed by the ascending or descending colon as the case may be, and of an irregular surface and consistence.

Sarcoma of the kidney is much more common. It constitutes a frequent form of abdominal tumor among children, especially the primary variety. The growth reaches a large size and may greatly distend the abdomen. It is almost always unilateral and presents objective characters similar to those of carcinoma, save that it is commonly less nodular and softer, in some cases even fluctuating over extensive or limited areas.

Hypernephroma, which may develop from suprarenal tissue exceptionally upon the kidney, as a rule within its substance, is a very common form of renal tumor. This neoplasm may be found upon post-mortem examination as small tumors within the cortex, or it may form large tumors having the characters of malignant growths of the kidneys in general. Metastases occur.

Symptoms of Malignant Tumors of the Kidney.—Pain, usually of a dull, dragging character and referred to the flank upon the affected side, is common. It may radiate to the thigh or extend to the pleura. There are large growths in which pain does not occur. Emaciation is commonly progressive and rapid, and a high grade of cachexia is usual. There are cases, however, especially of sarcoma, in which the nutrition and strength are fairly well maintained. Pressure upon the abdominal veins may cause venous distention and œdema of one or both lower extremities, and ascites. Pressure upon nerve-trunks may cause pain or pressure neuritis in the distribution of the intercostals or the crural nerves. Metastatic growths occur in various organs, and, in particular, in the lungs. They may occasion special symptoms. If the spinal cord is involved paraplegia may result.

Fig. 327.—Suprarenal sarcoma; metastases in the skull.—Robert Hutchinson.

The urine contains blood at some time in the course of a majority of the cases. The hæmaturia is intermittent, the urine being practically normal during the intervals. If the growth involves the pelvis the urine may be turbid and albuminous. The blood is sometimes fluid, sometimes clotted. It is characteristic of the hæmaturia of malignant disease of the kidney that blood-casts of the ureter, sometimes of the pelvis of the kidney, are occasionally passed. The passage of these clots is attended with intense pain like that of renal colic. In rare instances cancer elements have been discovered in the urine. Physical Signs.—If the growth involves a floating kidney, the organ may remain for some time movable and be found in the iliac fossa. When the kidney is affected *in situ* the tumor is stationary, unilateral, and develops from the upper and posterior region of the abdomen. Other attributes have been mentioned. Bimanual palpa-

tion is important. The relation of the colon as indicated by the tympanitic percussion sign which it yields is of great value in the differential diagnosis.

Diagnosis.—DIRECT.—The diagnosis of malignant tumor of the kidney in well-developed cases may be readily made when all of the above symptoms and signs enter into the symptom-complex. In proportion as several of them are absent the diagnosis becomes uncertain. The nature of the neoplasm cannot in all cases be positively determined. Carcinoma is more common in adult life, attended with a greater tendency to wasting and cachexia, and to hæmaturia. Sarcoma is far more common under ten years of age; it frequently runs its course without hæmaturia, and may be attended with little disturbance of the general health. If primary or metastatic growths accessible to direct examination are present, the determination of their character settles any diagnostic uncertainty as to the nature of the renal tumor. The examination of tissue elements found in the urine, or obtained by exploratory puncture, may yield conclusive results. The recent investigations of Kelly and others render it probable that a large proportion of the cases described as primary carcinoma and sarcoma of the kidneys are hypernephromata.

DIFFERENTIAL.—*Tumors of the Pelvic Organs.*—Tumors of the kidney are frequently mistaken for ovarian tumors. The greater mobility of the latter, their development from the pelvis, their relation to the uterus and the presence of a pedicle as determined by vaginal examination, and derangement of sexual functions, as menstruation, are significant. The presence or absence of intermittent hæmaturia is important. Tumors of the uterus are less likely to present diagnostic difficulties. *Retroperitoneal sarcoma*—Lobstein's cancer—may give rise to diagnostic uncertainty, particularly in children. Both conditions form very large tumors. The disease of the lymphatic glands is more central and less movable. It may extend to the kidneys. In the advanced stages the diagnosis cannot always be made.

The careful application of the methods of physical diagnosis in connection with the facts relative to tumors of the kidneys renders their discrimination from tumors of the liver and spleen an easy matter.

Prognosis.—The outlook is unfavorable. The extirpation of a small growth has in a low percentage of the cases been followed by recovery.

XII.

THE DIAGNOSIS OF DISEASES OF THE BLOOD AND THE BLOOD-MAKING ORGANS.

The bone-marrow is not directly accessible to clinical methods of examination, except in the case of the sternum and other flat bones, in which tenderness may occasionally be elicited by somewhat forcible palpation or percussion. Pathological changes in its substance are to be inferred usually from the abnormal findings in the morphology and numbers of the red and

white corpuscles corresponding to lesions found post mortem; very exceptionally from the presence of the Bence-Jones protein in the urine.

The superficial lymph-nodes, barely palpable under normal conditions, when enlarged in disease yield important direct physical signs upon inspection and palpation, while the deeper glands indicate their presence sometimes by great enlargement; sometimes by their pressure effects upon adjacent structures or organs. The surface contour, consistence, mobility, and so on are to be considered. In perisplenitis friction sounds may be detected upon auscultation and very rarely vascular murmurs. The blood-findings are also to be studied. The spleen falls easily within the scope of physical examination and yields upon inspection and palpation signs of diagnostic importance both in normal and abnormal conditions.

Enlarged lymph-glands may be punctured with the hypodermic needle and the fluid obtained examined for cellular elements, bacteria and parasites.

Puncture of the spleen has also been practised for diagnostic purposes, but this procedure is not without danger and the circumstances that would justify it are of extremely infrequent occurrence.

The excision of a superficial lymph-node under local anæsthesia may supply material for smears, catheters, animal inoculation and histological study. By this means a provisional diagnosis may be finally confirmed.

DISEASES OF THE BLOOD.

I. ANÆMIA.

Definition.—A morbid condition of the blood characterized by a diminution of the erythrocytes or the hæmoglobin, or of both.

i. General Considerations.

Pallor of the skin and mucous membranes is in a high degree suggestive of anæmia. For a positive diagnosis, however, a systematic microscopic examination is essential. By this method only can the existence of anæmia in every case be recognized and the nature of the anæmia positively determined.

Pseudo-anæmia.—There are individuals in whom marked pallor of the skin and mucous membranes suggest anæmia, but whose blood shows, upon microscopical examination, a normal number of erythrocytes and a normal percentage of hæmoglobin. Such persons usually owe their pallor to one or another of the following conditions: (1) hereditary peculiarities of the integuments, among which is an opaque but non-pigmented skin deficient in capillary network; (2) prolonged life in tropical regions, the so-called tropical anæmia; (3) chronic nephritis, arteriosclerosis, certain cases of cardiac disease, neurasthenia, and tuberculosis; (4) habitual indoor life, as in prisoners,—the so-called "prison-pallor,"—workers in sweat-shops, miners, and others whose occupations and circumstances deprive them of sunlight and fresh air.

There are transitory conditions in which pallor of the skin and mucous surfaces is ischæmic rather than anæmic, as syncope, rigor, chilling of the surface, fatigue, pain, and sudden intense emotions, especially fear. The

volume of the blood and its corpuscular values are unchanged, but it retires from the surface and accumulates in the viscera and deeper tissues of the body. The tidal blood flows and ebbs, not rhythmically, but under the influences of various physiological and pathological influences.

Local Anæmia.—The distribution of the blood mass is controlled by the arteries, which contract or dilate under the influence of the central or peripheral vasomotor ganglia. Hence afflux and deflux, physiological within limits; pathological in excess. In one territory congestion; in another anæmia. Cerebral anæmia with faintness or syncope results from sudden dilatation of the mesenteric vessels, such as is caused by intense emotion, pain, the rapid removal of pressure, as in the abrupt change to the upright posture, or a copious stool in advanced aortic incompetence, or the evacuation of a large ascites. Chronic anæmia of the central nervous system may be the cause of many of the vague symptoms in cardiac disease and enteroptosis. Local anæmias due to spasm of peripheral vessels, such as is seen in Raynaud's disease, causing asphyxia of the extremities, may affect visceral vessels, causing functional derangements, or circumscribed areas of brain tissue, and give rise to transitory palsies, aphasia, or hemicrania.

General Anæmia.—The general anæmias are primary, essential or cytogenic, and secondary or symptomatic.

ii. Primary Anæmia.

The blood-making organs are at fault. The etiology is obscure. The essential lesions directly involve the blood. Clinical phenomena manifest in other structures are secondary to changes in the blood. This group comprises chlorosis, pernicious anæmia, and splenic anæmia.

(a) CHLOROSIS.

Definition.—Anæmia of undetermined cause, common in females at or shortly after the age of puberty, and characterized by a peculiar greenish-yellow pallor of the skin, constipation, breathlessness upon exertion, and marked relative diminution of the hæmoglobin.

Etiology.—Predisposing Influences.—Sex is paramount. Chlorosis occurs only in females. The period of onset corresponds to that of puberty, and varies from about the thirteenth to the seventeenth year. Earlier than this period it is uncommon; later it is due to recurrences, which are sometimes multiple. The disease may occur in successive generations, and the daughters of mothers who had suffered from chlorosis are often chlorotic. Several girls in the same family often manifest the disease. The condition is sometimes associated with hypoplasia of the aorta and sexual organs. The disease occurs in every walk of life, but is much more common among the poorer classes and working girls in factory towns and large cities. It is not uncommon among domestic servants. Among farmers' daughters it is rare. Lack of exercise, of fresh air and sunshine, and insufficient and improper food are important predisposing influences. The subjects of the disease are often lethargic and phlegmatic; sometimes

emotional and nervous. The disease has, upon insufficient grounds, been regarded as a neurosis, attributed to copræmia from constipation and to the wearing of the corset.

The Exciting Cause.—The actual cause of chlorosis is unknown. It is apparently due to a functional default of the blood-making organs—defect of hæmogenesis—incident to the functional maturity of the reproductive organs.

Symptoms.—Pallor is marked, and the skin has a faint yellowish-green tinge to which is due the designation chlorosis, or green sickness. The conjunctivæ are faintly bluish and the mucous membranes of the mouth and lips very pale. In some cases the normal pigmentation about the folds of the joints is slightly intensified. The subcutaneous fat is frequently increased and there is an increased turgor of the integumentary tissues. There may be actual œdema of the face and ankles. There are rare cases in which the cheeks have a reddish color, especially upon exertion or during excitement—*chlorosis florida, chlorosis rubra.* Gastro-intestinal symptoms are common and often prominent. The appetite is capricious. The school-girl's fondness for pickles and bits of chalk is well known. Hyperacidity is often present and associated with epigastric distress. Dilatation of the stomach, gastroptosis, and movable right kidney are frequently present. Constipation is a common and troublesome condition. Dyspnœa, palpitation, and vertigo occur upon exertion, as in the rapid ascent of a flight of stairs. The heart is frequently dilated and the apex displaced slightly to the left. A systolic murmur in the mitral area may be the sign of relative insufficiency. Much more common are systolic murmurs at the base, particularly in the pulmonary area. Distinct pulsation in the second left intercostal space is not uncommon. Diastolic murmurs are infrequent. A distinct, loud, continuous, venous hum may be heard over the jugular vein on the right side—*nun's murmur, bruit de diable, humming-top murmur.* Pulsation may sometimes be seen in the veins of the neck; less frequently in the peripheral veins. Thrombosis may occur in the cerebral sinuses or in the left femoral vein. In the latter event there is danger of pulmonary infarct. Slight enlargement of the thyroid body is not uncommon. It may be associated with Joffroy's sign. Amenorrhœa and dysmenorrhœa are common. Hysterical manifestations occur in a large proportion of the cases. Headache, coldness of the extremities, and mental depression are prominent symptoms. Moderate rises of temperature are occasionally observed.

The Blood.—The droplet is pale and flows freely from the puncture. It is characterized by transparency and fluidity as contrasted with normal blood. The coagulation period is short and the specific gravity decreased. The alkalinity of the blood, according to most observers, remains normal. There is marked absolute decrease in the hæmoglobin; and a high degree of diminution in hæmoglobin relative to the reduction in the number of erythrocytes, as manifested by low color indices, constitutes a characteristic phenomenon of chlorotic blood. Microscopically the erythrocytes are moderately decreased, usually to about 4,000,000 per cubic millimetre. They may fall as low as 3,000,000 or even 2,000,000 in severe cases. There is a slight general diminution in their average diameter. Nucleated forms

—*normoblasts*—are occasionally present. Poikilocytosis is common and may be marked in severe cases. Microcytosis also occurs in the graver cases. Polychromatophilia is rare. The leucocytes are usually normal or slightly increased. There is relative increase in the number of lymphocytes. Small percentages of myelocytes occur in severe cases. Eosinophiles are absent in the majority of the cases.

Pseudochlorosis.—This term has been applied to a rare condition in which the characteristic symptom-complex is present, but no marked change in the blood can be discovered upon ordinary clinical examination. It has been assumed that the actual condition of the blood as regards the relation between the hæmoglobin and erythrocytes is masked by a diminution in the volume of plasma.

Diagnosis.—The DIRECT DIAGNOSIS of chlorosis is unattended with difficulty. The pallor, the pearly or bluish conjunctivæ, the preservation of subcutaneous fat occurring in a girl at or shortly after puberty constitute a characteristic clinical picture. Headache, breathlessness upon exertion, and gastro-intestinal symptoms, especially constipation, are of diagnostic value, particularly in the absence of visceral disease attended by anæmia and dropsy, as disease of the heart, or forms of nephritis, or chronic infections, as tuberculosis, syphilis, or malaria. In the majority of cases the characters of the blood differentiate chlorosis from other forms of anæmia; but the fact is not to be overlooked that there are cases of secondary anæmia, particularly in the above-mentioned organic and infectious diseases, in which changes in the blood closely corresponding to those of chlorosis occur.

DIFFERENTIAL DIAGNOSIS.—*Cardiac Disease.*—The dyspnœa and palpitation upon exertion frequently suggest valvular disease of the heart. The anamnesis is important. A history of rheumatic or scarlet fever, followed by such symptoms becoming progressively more severe; distinct cardiac enlargement; murmurs having characteristic points of maximum intensity, definite lines of propagation, and constant relations to the revolution of the heart; the absence of a venous hum, are in favor of disease of the heart. The sex and age of the patient are highly important. *Renal Disease.*—General pallor and some degree of anasarca, as shown in puffiness of the face and œdema of the feet, may be suggestive of nephritis—a diagnosis at once negatived by the absence of albumin and casts. The possibility of transient albuminuria—*albuminuria of adolescence*—is to be borne in mind. The intermittent nature of this form of albuminuria and its tendency to disappear upon rest are significant. *Tuberculosis.*—The pallor of the early stages of pulmonary tuberculosis in a young girl may simulate chlorosis. Cough, wasting, positive physical signs, rapid pulse, and a slight rise of temperature recurring about the same time every day, increased by exercise and at the time of menstruation, point to phthisis. The examination of the blood is essential. The deficiency of the hæmoglobin in chlorosis may be apparent when a drop of blood is allowed to fall upon a piece of white blotting paper or a handkerchief, the blood of a healthy person being used for contrast. *Malaria.*—The character of the febrile paroxysm and the presence of malarial parasites in the blood are conclusive. *Syphilis.*—The rapid anæmia and the fever of secondary syphilis

may give rise to doubts as to the diagnosis. The macular syphilide, adenopathy and mucous patches are diagnostic. When a chlorotic girl contracts syphilis the question of diagnosis becomes complicated.

Prognosis.—The prognosis is favorable. The majority of cases recover under treatment in the course of a few weeks. The tendency to repeated attacks persists in some cases for several years. The influence of pregnancy and lactation in arresting this tendency is usually positive. The administration of iron in proper doses appears in a majority of cases to exert a specific curative influence—a fact that might be of diagnostic importance in a doubtful case.

(b) PERNICIOUS ANÆMIA.

Idiopathic or Progressive Anæmia. Chronic Hemolytic Anæmia.

Definition.—Severe anæmia developing either idiopathically or in the absence of discoverable adequate cause, and characterized by a progressively unfavorable course, maintenance of body weight, and constant changes in the blood, namely, great reduction in the number of erythrocytes, megalocytosis, microcytosis, poikilocytosis, the presence of erythroblasts, and relative increase in hæmoglobin.

Etiology.—Clinically several different groups of cases may be recognized which present the symptoms of progressive pernicious anæmia, but are due to different causes. A majority of the cases correspond to the description of Addison, and arise in the absence of the usual causes of anæmia. Intense progressive anæmia, presenting all the clinical features of pernicious anæmia, is occasionally encountered, (a) in child-bearing women, beginning either during pregnancy or after parturition; (b) in gastro-intestinal diseases, particularly atrophy of the stomach; and (c) in certain forms of intestinal parasitism, especially uncinariasis and the presence of the Bothriocephalus latus. The blood picture seen in the anæmia which follows certain cases of nitrobenzol poisoning is similar to that of pernicious anæmia. Cases have been observed in every quarter of the globe. The onset is gradual and wholly independent of seasonal influences. The exacerbations which follow remissions under treatment sometimes occur in the spring of the year. The onset of the disease is most common in late middle life. It has been observed at all ages. Excluding the cases which begin during pregnancy and lactation, it would appear that males are much more frequently affected than females. Rare cases have been encountered in parents and children—a fact which does not warrant the assumption that the tendency to the disease is transmitted from the parent to the offspring. Severe nervous or mental shock and prolonged intense depressing emotions have in many instances been followed by the onset of the disease. The researches of William Hunter lend support to the conclusion that this form of anæmia is due to chronic septic infection associated with lesions of the gums or mouth, and gastric and intestinal sepsis, which lead to hæmolysis.

Symptoms.—The onset is insidious, and the symptoms are those of a profound and progressive anæmia. Pallor of the skin and visible mucous

membranes, languor, breathlessness upon slight exertion or emotional excitement, flabbiness of the tissues associated with remarkable preservation of the subcutaneous fat and slight puffiness about the ankles, extreme debility, make up the clinical picture. In a majority of the cases there is irregular fever of moderate intensity. Cardiovascular symptoms consist of faintness, dyspnœa and palpitation upon exertion, a full, soft, and compressible pulse, visible pulsation of the superficially placed arteries, and hæmic murmurs. The pulse not rarely resembles the water-hammer pulse of aortic regurgitation,—*Corrigan's pulse,*—a resemblance frequently increased by the presence of capillary and a penetrating venous pulsation. There is a marked tendency to hemorrhage from the mucous membranes and into the skin. Retinal hemorrhages are common as in other grave anæmias. *Gastro-intestinal Symptoms.*—The appetite fails, and in advanced cases anorexia is complete. The mouth is dry, the tongue furred, often sore, the gums are ulcerated, and the breath is offensive. Nausea and vomiting are common. Achlorhydria is almost always present and in some cases achylia gastrica. Attacks of diarrhœa occur without apparent cause. The urine is abundant and usually of low specific gravity; sometimes pale, sometimes of a deep sherry color, due to the presence of urobilin. The skin is blanched, smooth and waxy in appearance. It is commonly intensely pale, frequently of a faint lemon color, especially marked upon the hands, sometimes subicteroid, and less commonly pigmented as in Addison's disease. The pigment may be more or less uniformly distributed, or deposited in irregular patches. It is sometimes associated with patches of vitiligo. In some cases the cutaneous pigmentation follows the prolonged administration of arsenic.

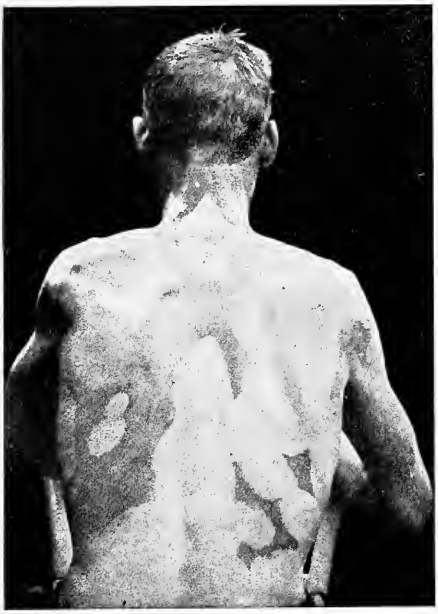


Fig. 328.—Pigmentation of the skin in a case of pernicious anæmia.—Packard.

Spinal symptoms may be encountered as the result of degeneration involving the posterior and lateral columns, and in some cases extending beyond these tracts to the anterior part of the cord. They may appear earlier than the blood changes, but more commonly not until the disease is well advanced. They consist of numbness and tingling in the legs and feet, weakness, and in some cases severe pain. The reflexes are increased. Later similar symptoms may involve the upper extremities. There may be marked ataxia, with disturbance of the functions of the bladder and rectum. After a time loss of sensation may occur, with flaccidity and abolition of the reflexes.

The Blood.—There is absolute diminution of the hæmoglobin, which is, however, increased relatively to the number of red cells, so that the

color index is high. The erythrocytes show a great numerical decrease, often to 1,000,000, or in terminal states to 500,000 per cubic millimetre. Erythroblasts are constant, the common form being megaloblastic. Poikilocytosis is constant and marked. Megalocytes and microcytes occur, the former being more common and more marked. Polychromatophilia is found in many of the erythrocytes, both non-nucleated and nucleated. The leucocytes are usually decreased, often markedly so. A relative increase in the lymphocytes is common. Myelocytes are usually present. The eosinophiles are almost always decreased in number and frequently absent altogether. The number of blood-plaques is variable. Blood crises (von Noorden) are characterized by the appearance in the blood of large numbers of nucleated red blood-corpuscles, very often in successive crops, and are usually of sudden onset and brief duration, sometimes lasting but a few hours. These crises are common in severe anæmia following hemorrhage and in chlorosis, and not rare in some forms of leukæmia and in pernicious anæmia, and are usually followed by periods of temporary improvement in the blood count. The blood crises of pernicious anæmia are more commonly of the megaloblastic type and are frequently followed by the death of the patient. When of the normoblastic type they may be followed by an actual increase in the erythrocytes.

Aplastic Anæmia.—This term has been applied to a limited group of cases presenting the symptoms of pernicious anæmia but characterized by atrophy of the erythroblastic tissue in the bone-marrow. Clinical differences between this variety and the ordinary form of pernicious anæmia are found in its earlier occurrence, a majority of the cases having occurred before the thirty-fifth year of life; its greater frequency among females than males; its rapid course, unattended by remissions and usually terminating within a period measured by months; a greater tendency to hemorrhage. Differences in the blood picture consist in a lower color index than in the ordinary form; a marked increase in the percentage of lymphocytes; the absence, as a rule, of erythroblasts; the absence or comparative infrequency of poikilocytosis, anisocytosis, polychromatophilia; and the great diminution of the blood-plates. Pathologically the most constant and striking change is manifest in the marrow of the long bones, from which the erythroblastic tissue has disappeared, leaving the medullary cavities filled with fat. That this change has occurred in all the bones has not as yet been demonstrated in any case. The femur is used as a standard, and if its marrow is yellow and homogeneous throughout, the form of anæmia may be regarded as aplastic. *Diagnosis.*—The diagnosis depends upon the absence of regenerative forms in the blood on repeated examinations. It is rendered probable by the concurrence of the above clinical symptoms and hæmatological findings, but finally rests upon the post-mortem examination of the marrow of the bones.

Diagnosis.—The direct diagnosis of pernicious anæmia can only be made by a microscopical examination of the blood. In general practice the true nature of the disease is not often suspected in its earlier course and rarely recognized after it has made some progress. Even the blood changes are not at all times present in every case in the beginning. A severe anæmia insidiously arising in the absence of any obvious cause,

pursuing an unevenly progressive course but little influenced by treatment; preservation of the subcutaneous fat and body weight to a remarkable degree; a blanched, smooth, and waxy appearance of the surface, which has a faint lemon-yellow tint; extreme languor and breathlessness upon exertion; a tendency to hemorrhage into the skin or from mucous surfaces; retinal hemorrhage; gastric symptoms; and the occurrence of febrile paroxysms of moderate intensity constitute a symptom-complex which is highly characteristic. A blood picture showing the association of oligocythæmia of high grade, falling in many cases below 1,000,000 per cubic millimetre; erythroblasts mostly of the megaloblastic type; poikilocytosis; great variation in the size of the erythrocytes; a high color index; and leukopenia render the diagnosis positive.

Differential.—1. *Grave secondary anæmias,* such as occur after copious hemorrhages (especially a prolonged habitual blood loss), visceral cancer (especially carcinoma ventriculi), and in advanced syphilis, often present clinical symptoms precisely similar to those of pernicious anæmia. The anamnesis is important. As a rule, an obvious cause for the anæmia may be discovered. There are, however, cases in which malignant disease cannot be located, or the history of syphilis is obscure. The actual pathological condition then rests upon the examination of the blood. The following points are of diagnostic importance: In secondary anæmia, (a) the oligocythæmia is less marked, the count rarely falling below 1,000,000 per cubic millimetre; (b) the color index is lower; (c) leucocytosis is often present; (d) megalocytosis does not occur. The fault does not primarily involve the hæmatopoietic organs. The blood changes result from, and are secondary to, constitutional diseases, as certain of the acute and chronic infections and intoxications, diabetes, parasitism and nutritional disorders, local anatomical lesions which seriously interfere with the functions of important viscera,—as the heart, lungs, the organs of digestion, or the kidneys,—malignant disease, or to hemorrhage. The anæmia is due to deficient blood formation—hæmogenesis; excessive blood destruction—hæmolysis; or the association of these two processes. Severe secondary anæmia, when long continued, may exhaust the function of the blood-making organs in such a manner as to be converted into primary anæmia. 2. *Chlorosis.*—There are often clinical phenomena present which suggest pernicious anæmia, namely, preservation of subcutaneous fat, smooth, waxy, and faintly greenish-yellow tint of the skin, pallor of mucous surfaces and pearly tint of sclera, breathlessness, and languor; but the blood examination shows essential differences. The following points are to be considered: (a) In chlorosis we have to do with a hæmoglobin rather than a corpuscular anæmia; hence a low color index, the reverse of the condition in pernicious anæmia; (b) there is a general diminution in the diameter of the erythrocytes; (c) megaloblasts may occasionally be encountered, but are never present in great numbers—nucleated red corpuscles when seen are almost always normoblasts; (d) leukopenia of high grade is not common; (e) myelocytes may be observed in small numbers in both diseases, but are much less common in chlorosis. 3. *Bothriocephalus Anæmia.*—The therapeutic test yields positive results. Metabolic products of the intestinal parasite have been thought to possess

hæmolytic properties. Its expulsion may be promptly followed by an improvement in the general health and the restoration of the blood to its normal condition, the megaloblasts and oligocythæmia disappearing and the color index falling to the usual range of health. 4. *Various Affections.*—In the absence of a blood examination the lemon-yellow tint of the skin may suggest jaundice, from which pernicious anæmia is to be differentiated by the pearly sclera, the fact that bile pigments are not present in the urine, and the absence of definite signs and symptoms indicating disease of the liver or bile passages; the anæmia, pufliness of the face, and swelling of the ankles may simulate nephritis, especially if, as is sometimes the case, albumin is found in the urine, a view not sustained by the results of close analysis of the urine and the history of the case; palpitation, dyspnœa, and the condition of the arteries point to cardiac disease, but the anamnesis and physical signs lend little support to such a diagnosis; finally, nervous symptoms, such as numbness of the legs and feet, less commonly of the hands, pain, sometimes very severe, impairment of station and gait, and loss of the deep reflexes arouse the suspicion that the affection is of spinal origin, a diagnosis always obscure but much influenced by the blood examination, since pernicious anæmia has not been found to arise as a secondary affection in spinal degenerations, while posterolateral sclerosis has been frequently observed in this form of anæmia.

Prognosis.—The course of the disease is not, as a rule, steadily progressive. There are periods of improvement followed by relapse. Many cases go on in this way for years. There are cases which run a very rapid course and end fatally within a few months. The average duration is about a year or fifteen months. Occasionally recoveries have been recorded. The following conditions are of bad omen: an oligocythæmia of less than 1,000,000 per cubic millimetre, a high percentage of megaloblasts and blood crises of megaloblastic type, the inability to take arsenic, tendency to hemorrhage, gastro-intestinal disturbances, and, as in almost all grave chronic diseases, the privations and disabilities incident to poverty.

(c) SPLENIC ANÆMIA; BANTI'S DISEASE.

This affection is considered under the heading Diagnosis of Diseases of the Spleen.

iii. Secondary or Symptomatic Anæmia.

Under this heading are included those forms of anæmia caused by acute and long repeated hemorrhages, certain intestinal parasites, unhygienic surroundings, insufficient food, prolonged lactation, the metal poisonings, acute and chronic infections, acute and chronic visceral diseases, especially nephritis and cardiac disease, and malignant growths.

In moderate cases the freshly drawn blood presents an appearance nearly normal, but in cases of intense secondary anæmia it may look like serum faintly tinged with crimson. In the latter case the tendency to rouleaux is slight. The coagulation period is diminished in proportion to the intensity of the anæmia. The further changes in the blood are as

follows: hæmoglobin diminished to an extent proportionate to the energy of the cause; color index correspondingly decreased; erythrocytes diminished to a varying degree; nucleated forms in intense anæmia, the normoblastic type prevailing; departures from normal in the size and shape, and polychromatophilia in severe cases; leucocytosis commonly present; polynuclear neutrophiles relatively increased; lymphocytes decreased; lymphocytosis may occur in severe and prolonged cases; myelocytes in small numbers; blood-plaques increased. The coagulation time is shortened.

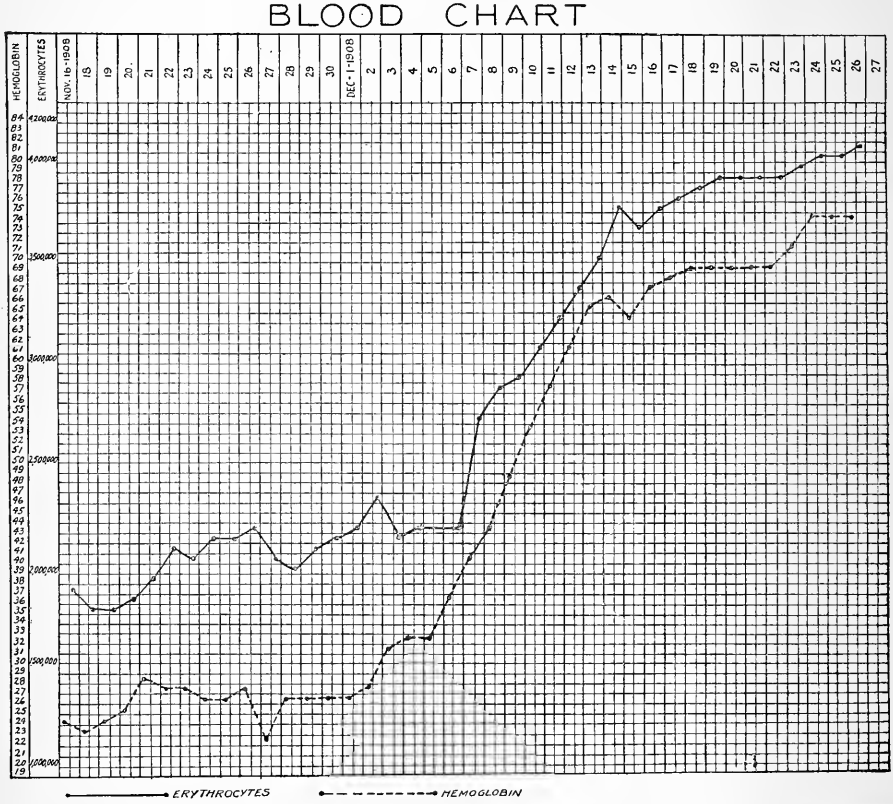


Fig. 329.—Chart showing variations in the number of erythrocytes and in the percentage of hæmoglobin in posthemorrhagic anæmia due to gastric ulcer.

Acute Posthemorrhagic Anæmia.—An examination directly after the blood loss may fail to show diminution in the hæmoglobin or corpuscles by reason of the oligæmia. Fluid is, however, rapidly taken from the tissues into the capillaries so that a condition of hydræmia ensues and the decrease in hæmoglobin and erythrocytes becomes apparent, the minimum counts occurring some time within the course of a week. A more or less marked leucocytosis commonly occurs and persists for about a week, gradually declining. A gradual regeneration of the blood takes place in uncomplicated cases and is completed in from three to four weeks, the hæmoglobin reaching normal at a later period than the erythrocytes.

The normal amount of blood is about one-twentieth of the body weight.

The sudden loss of one-half his blood in an adult usually proves fatal. A much smaller loss may cause the death of a child. Death under these circumstances results in part from insufficient oxygen; in part from the sudden rapid fall of arterial pressure. The first of these deficiencies may be met by transfusion of blood; the second by the intravenous injection of normal salt solution. The symptoms are pallor, faintness, syncopy, dyspnœa, and finally collapse.

Chronic Post-Hemorrhagic Anæmia.—Anæmia caused by continuous small blood losses or small hemorrhages repeated at short intervals for a considerable time.

The blood picture is that of the acute form of post-hemorrhagic anæmia. The gradual development permits of an anæmia of very high grade, the erythrocytes in some cases falling below 1,000,000 and the hæmoglobin to 25 per cent. or even lower.

The symptom-complex comprises progressive pallor, weakness, loss of energy, dyspnœa, and palpitation upon moderate exertion and mental hebetude.

The common causes are gastric and duodenal ulcer, hemorrhoids, recurrent bleeding at the nose, hook-worm disease, amœbic dysentery, chronic metrorrhagia and bleeding in various conditions arising in hæmophilia and the hemorrhagic diseases.

Secondary anæmia is very common in childhood. It may be congenital, as in syphilis and other infections, or acquired, as in (a) hemorrhages of various kinds, particularly from the navel, after circumcision, and in the purpura group, or (b) from general causes, as malnutrition, improper hygiene, syphilis, rickets, tuberculosis, the fevers, sepsis, gastro-intestinal and other visceral diseases, nephritis, acute disease of the heart, and malignant disease.

II. POLYCYTHÆMIA RUBRA.

A condition of the blood, characterized by a persisting increase in the number of the red blood-corpuscles. There are two groups of cases: (a) those in which the increase is symptomatic—the *erythrocytes:* and (b) those in which it is due to a derangement of the blood-making function of the bone-marrow—*erythræmia.*

(a) The Erythrocytoses.

Definition.—Erythrocytosis is a more or less permanent increase of the red blood-corpuscles of unknown causation which occurs in certain cases of chronic dyspnœa and among persons living at a high altitude.

Etiology.—Predisposing Influences.—The chronic shortness of breath in which this condition occurs may be due to congenital heart disease, especially pulmonary stenosis; or to myocardial insufficiency or laryngeal or tracheal stenosis or advanced pulmonary emphysema. The condition occurring in the absence of these affections in persons living at a high altitude is due to causes not well understood.

The Exciting Cause.—Theoretically over-stimulation of the blood marrow to meet the requirements of a deficient oxygen supply has been assigned as a cause.

Symptoms.—The symptom-complex of the underlying disease is present. Cyanosis is often marked. The *morbus cæruleus* of congenital heart disease is not always attended by an increase of the red corpuscles. Hyperplasia of the red marrow has been found. The red-corpuscles in the congenital disease may reach 9,000,000 per cmm.; in acquired myocardial disease they rarely reach 7,000,000; in respiratory stenosis the number is scarcely so high and in the high altitude form it is usually about 5 to 7,000,000.

Diagnosis.—To what extent a derangement in the distribution of the red corpuscles, or a general increase in their numbers per cmm. or concentration is at fault cannot always be known. An increased production is accompanied by nucleated reds and polychromasia. It is important to differentiate between the transient increase of the reds occurring immediately after the ascent to a high altitude and the permanent erythrocytosis arising during a prolonged sojourn at such a level.

(b) Erythræmia.

Osler's Disease.

See Splenic Tumor with Polycythæmia and Cyanosis.

III. LEUKÆMIA.

Definition.—Leukæmia is an affection of undetermined causation characterized by the presence in the blood of abnormal leucocytes in great but fluctuating numbers, with an increase in the normal forms, with a decrease in the erythrocytes and hæmoglobin, together with proliferation of the lymphadenoid or the myeloid tissues of the body.

Varieties.—Two well-defined clinical varieties are recognized: (1) myeloid leukæmia, and (2) lymphadenoid leukæmia. The myeloid is much the more common type.

Etiology.—Predisposing Influences.—The disease has been encountered at all periods of life, from early infancy to the seventieth year. It is most common in the third, fourth, and fifth decades. Leukæmia is more common in males than females in the ratio of about two to one. Cases have been observed in pregnancy, and the disease not rarely develops at the grand climacteric. There are instances in which leukæmia has been observed in successive generations, and a leukæmic mother has borne leukæmic children. On the other hand, leukæmic mothers have borne non-leukæmic children, and a woman showing no signs of the disease has borne a leukæmic child. Leukæmia occurs in all parts of the world. It is not rare in the United States. Cases have been observed in various domestic animals, especially fowls and dogs.

Exciting Cause.—The disease has upon inadequate grounds been attributed to malaria. It has followed blows and grave bodily injury. The tendencies to hemorrhage and to habitual nose-bleeding, which have been regarded as etiological, are early manifestations of an affection the true nature of which has been revealed by a study of the blood. Nothing is as yet known of the actual cause. Organisms of the diphtheroid type occur in the lesions. The presence or history of a focal lesion in the upper respiratory tract—tonsils, adenoid tissue, nose, accessory sinuses, bronchi or in

connection with the teeth or of severe pyogenic processes elsewhere—lend support to the hypotheses of an infectious origin.

General Symptoms.—The onset is insidious and may be associated with persistent and intractable gastro-intestinal symptoms. In some instances the patients present the appearance of fairly good health until the occurrence of grave symptoms shortly before death. A lad of sixteen was supposed to be in his usual health until the occurrence of hemorrhage from the stomach, which proved fatal in the course of two days. Such cases are not very uncommon. Epistaxis is a frequent symptom. Pallor, palpitation, and dyspnœa upon exertion are very common. Distention of the abdomen due to splenic tumor and enlargement of the liver may first attract the attention of the patient. Tenderness over the sternum or the long bones occurs in many of the cases. Diffuse enlargement of the superficial lymph-nodes, which are generally of moderate consistency, not adherent among themselves or to the skin, and variable in size from time to time, is common in the lymphatic form. The groups usually involved are the cervical, axillary, and inguinal. These changes may be restricted to the mesenteric and other deep groups without demonstrable signs of enlargement during life, and there are cases in which the bone-marrow alone is involved, without enlargement of the spleen or lymphatic glands. There may be a high degree of emaciation and anasarca, or effusion into the serous sacs. Acute and chronic forms occur.

Much confusion exists in the nomenclature and classification of the leukæmias. Recent studies indicate a disposition among pathologists to assume a common etiological or general relationship in the group of diseases comprising the leukæmias, pseudoleukæmia, lymphosarcoma, malignant lymphoma, chloroma, leukosarcoma and Hodgkin's disease.

i. Myeloid Leukæmia.

Myelogenous Leukæmia.

Symptoms.—Gradual enlargement of the spleen is usually the most conspicuous clinical phenomenon. The enlarged organ extends downward and to the right, and may reach the level of the pubic arch and pass beyond the median line. Its well-defined border and often the notch or notches may be distinctly felt. It is usually painless, but occasionally both pain and tenderness are present. Perisplenic friction may, in such cases, be recognized upon auscultation and palpation. Minor fluctuations in size may be observed: enlargement during digestion, and diminution during fasting and after diarrhœa or free hemorrhage. Fluctuations of greater amplitude frequently occur as spontaneous events in the natural history of the disease, or in consequence of the administration of arsenic or other therapeutic measures. Massive splenic enlargement causes pressure symptoms, among which the more common are distress after eating and constipation. Gastro-intestinal symptoms occur in the course of almost every case. Nausea and vomiting may be early and persistent. Diarrhœa is common. Hemorrhage from the bowel is not common. It may be due to chronic dysentery. Fatal obstruction of the bowel may be caused by pressure of the enlarged spleen upon the intestine. Jaundice is a rare symptom. Peritonitis and ascites may also be caused by the pressure of a massive

spleen. Symptoms due to circulatory derangements and the changes in the blood are prominent. The cardiac impulse is displaced upward an interspace or more by the big spleen; hæmic murmurs may be heard. The pulse is usually of large volume and rapid, but soft and compressible. The dyspnœa is anæmic. Hemorrhage is a very common symptom. Epistaxis, bleeding from the gums, hæmatemesis, purpura, retinal hemorrhage, and cerebral hemorrhage are frequent. Hæmoptysis and hæmaturia are much less common. Fluid exudates into the serous sacs are usually hemorrhagic. Œdema of the feet and general anasarca are terminal conditions. As in all the grave anæmias, headache, vertigo and syncopal attacks are of frequent occurrence. The lymph-nodes and liver may be somewhat enlarged by the presence of leucopoietic deposits of myeloid tissue. The bones, especially the sternum, ribs and the tibia, may be tender upon pressure. Irregular fever and night sweats occur. Leukæmic retinitis may be due to hemorrhage or minute leukæmic deposits. Optic neuritis is rare. Deafness is common and the syndrome known as Menière's disease has been observed. With the exception of a constant excess of uric acid the urine presents no characteristic changes. Priapism has been frequently noted. It may be an early and persistent symptom. Pneumonia or pulmonary œdema are common terminal events.

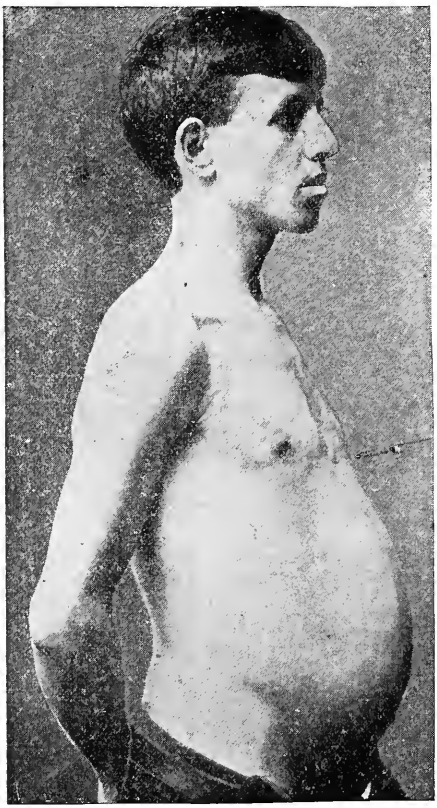


Fig. 330.—Myeloid leukæmia; distended abdomen due to massive enlargement of the spleen.—Jefferson Hospital.

The Blood.—The hæmoglobin is diminished not rarely as much as 50 per cent. The color index is correspondingly low. Exceptionally the color index is high.

Diminution in the number of erythrocytes is constant but not extreme. Counts falling below 2,000,000 are rare. Nucleated erythrocytes are very numerous, the normoblastic type being in excess. Megaloblasts are relatively less numerous and frequently present in atypical forms. Deformities in size and shape are present in varying degrees, dependent upon the extent of the hæmoglobin loss and cellular diminution, together with polychromatophilia affecting both the nucleated and non-nucleated erythrocytes.

The leucocytes are enormously increased. Counts of 100,000 per cubic millimetre are common, of 200,000 by no means rare, and of 500,000 occasional. When the oligocythæmia is marked the whites may nearly equal or even exceed the reds. The number flutuates widely from time to time in the course of the disease, and frequently undergoes remarkable diminu-

tion under the free administration of arsenic. Wide diurnal fluctuations are observed in some cases, the lower counts being noted late in the day. Extreme changes occur in certain cases, both in the blood picture and in the general condition, so that periods arise in which there are neither symptoms nor signs of leukæmia present. These remarkable remissions are not, however, permanent. Such reversions to the normal blood conditions accompanied by involution of the splenic tumor have frequently followed the therapeutic use of the Röntgen rays. Myelocytes in great numbers are present and constitute at least 20 per cent. of all leucocytes, as determined by the differential count in the majority of the cases. The polymuclear neutrophiles are much increased, but their percentage is relatively low. Many of these cells are dwarfed and deformed. The relative percentage of

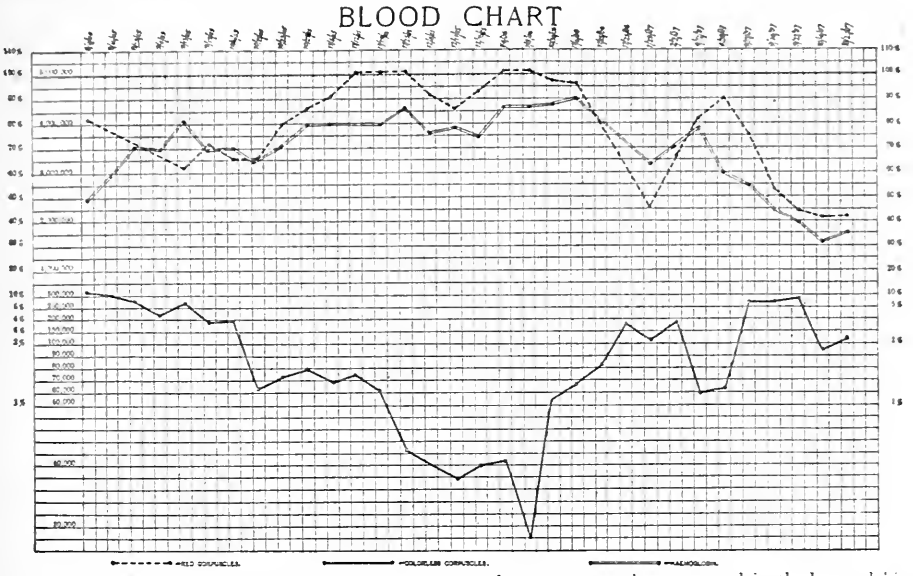


FIG. 331.—Chart showing variations in the number of leucocytes, erythrocytes, and in the hæmoglobin percentage in a case of myeloid leukæmia.

lymphocytes is much below normal. Eosinophilia is almost invariably present. In the majority of cases mast-cells are present in considerable numbers and the blood-plaques are much increased. Notwithstanding the remissions that occur the disease is incurable. Its average duration is about three to five years. The autopsy shows diffuse proliferation of myeloid leukopoietic tissue, involving the red marrow, the marrow of the long bones, the lymph-nodes, the liver and kidneys, the mucous and serous membranes, and this is found to be the chief cause of the massive enlargement of the spleen.

There is a rare acute form attended by the features of an acute malignant infection.

ii. Lymphadenoid Leukæmia.

Lymphatic Leukæmia.

Symptoms.—The general symptoms are similar to those of the myeloid form. There is enlargement of the lymph-nodes, which may involve the superficial or merely the deeper groups and is usually associated with more

or less marked enlargement of the spleen. The glands of the neck are commonly the most conspicuous, but those of the axillæ and groins are also enlarged. They are never of great size and can be moved under the skin. Usually they are sensitive to pressure. In the acute cases the lymph structures of the mouth, tongue, and pharynx may be enlarged. Enlargement of the thymus gland is sometimes present in this group of cases and may be the cause of characteristic symptoms: stridulous respiration with dyspnœa, usually paroxysmal.

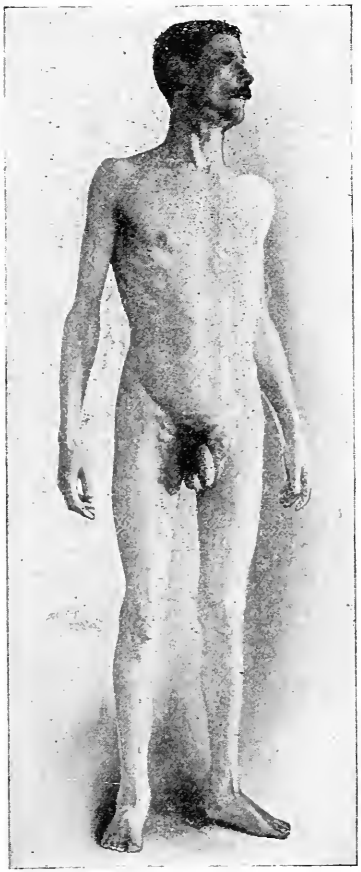


Fig. 332.—Chronic lymphatic leukæmia. —Jefferson Hospital.

The Blood.—Hæmoglobin is much diminished and the color index correspondingly low. The erythrocytes are reduced in number to 3,000,000 per cubic millimetre or lower. Nucleated red cells are scanty, those of the normoblastic type predominating. Deformities in size and contour and the tinctorial reactions are proportionate to the degree of degeneration of the blood—anæmia. The leucocytes are much increased in number, but not to the extent often seen in myelogenous leukæmia, counts above 200,000 being comparatively unusual. The differential count shows this increase to be largely due to an excess of large and small lymphocytes, which comprise about 90 per cent. of all forms. There are numerous atypical forms. The relative proportion of polynuclear neutrophiles is much reduced, those cells not usually exceeding 5 or 10 per cent. of all forms. Myelocytes are present in almost all cases, but in very small numbers. Eosinophiles are also present in the majority of the cases in scanty numbers. The blood-plaques may be diminished in number. The coagulation time is prolonged.

In lymphadenoid leukæmia also there is an acute form which, however, is of infrequent occurrence. It is seen most commonly in children and young adults. The course is almost invariably fatal, the duration not exceeding a few weeks. The actual cause is unknown.

The onset is abrupt with chill, high fever, and the symptoms of a severe stomatitis. The gums become spongy and bleed and this is followed by a general hemorrhagic diathesis. The tonsils are swollen and often covered with a grayish exudate. There is albuminuria with casts. The spleen becomes slightly enlarged. As the disease progresses a general hyperplasia of the lymph-nodes takes place.

The blood picture is at first inconclusive but presently the signs of an acute leukæmia appear. The red corpuscles, at first normal, diminish in

numbers until there is a condition of marked anæmia. The hæmoglobin is diminished; the color index below 1. The white corpuscles show an enormous increase in a short time of 100,000 or even 500,000, and are nearly all of the lymphocytic type.

The autopsy shows diffuse hyperplasia of the lymphadenoid tissues of the body.

Leukanæmia.—Lenbe has used this term to designate a symptom-complex consisting of intense anæmia with changes in the form of both the erythrocytes and leucocytes—conditions sometimes described as transitional or mixed forms of leukæmia and pernicious anæmia. He expressly reserves this term for forms of blood disease in which "both leucocytes and erythrocytes are uniformly and decidedly damaged in their development, and the case can neither be put in the category of leukæmia nor in that of pernicious anæmia." The cases are of sudden onset, with signs suggestive of an acute infectious process. In some of the cases there is severe tonsillar angina; in others stomatitis, extreme pallor, weakness, fever, hemorrhages, hyperplasia of the spleen, and moderate enlargement of the liver. Enlargement of the lymph-nodes is not constant. The course is unfavorable; the duration varies from a few days to several weeks.

Chloroma.—The blood changes in this rare affection are similar to those of lymphatic leukæmia. There is progressive diminution of the hæmoglobin and erythrocytes, associated with increase in the leucocytes. Deformities in size and shape in the erythrocytes, and nucleated forms, chiefly normoblasts, show themselves as the anæmia becomes more intense. There is marked lymphocytosis. The symptom-complex consists of progressive weakness, pallor, orbital pain, exophthalmus, deafness, and elastic swellings in the orbital and temporal regions. There is irregular hyperplasia of the bone-marrow with subperiosteal infiltrations and tumor-like metastases of greenish color—so-called "green cancer." The blood condition closely resembles acute lymphatic leukæmia, and chloroma has been regarded as a malignant form of leukæmia with greenish infiltrations and metastases.

Diagnosis.—The direct diagnosis of leukæmia can only be made by a microscopical examination of the blood. The disease is always leukæmia when the proportion of leucocytes to erythrocytes is 1 to 15 or less; when the leucocyte count is more than thirty times greater than normal and when at the same time many of the cells are conspicuously immature—myelocytes, large lymphocytes, and nucleated red corpuscles.

Even in cases in which there is a temporary return to the normal ratio between the leucocytes and the erythrocytes, immature forms very rarely seen in normal blood—erythroblasts and myelocytes—are present in sufficient numbers to warrant a provisional diagnosis of leukæmia in the absence of a previous knowledge of the conditions of the blood.

Differential.—The discrimination between myeloid and lymphadenoid leukæmia can only be made by the blood examination. In the myelogenous form the type of the blood is myelocytic, that is to say, myelocytes are present in enormous numbers together with an increase in the eosinophiles and mast-cells; oligocythæmia is moderate, erythro-

blasts are numerous, the normoblastic type predominating. In the lymphatic form the blood type is lymphocytic, namely, there is an excessive increase in the lymphocytes, myelocytes being absent, or present in very scanty numbers; there are very few eosinophiles or mast-cells; oligocythæmia is marked; and erythroblasts are few in number and proportionate to the general deterioration of the blood.

The clinical manifestations as regards the spleen and superficial lymph-nodes do not afford a basis for the differential diagnosis between the two forms of leukæmia; but this fact is without importance since an examination of the blood is essential to the general diagnosis.

Hodgkin's Disease.—The enlargements of the lymph-nodes and of the spleen in this affection often closely simulate leukæmia. The differential diagnosis rests upon the fact that the blood is normal or presents the changes which occur in the various forms of secondary anæmia. Associated inflammatory or infectious processes when present may cause an increase of leucocytes with the characters of a polynuclear neutrophile leucocytosis.

Chloroma may be recognized by the exophthalmus, orbital pains, and elastic tumor formations.

Splenic Anæmia.—The remarkable size of the splenic tumor, absence of enlargement of the superficial lymph-nodes, and a high grade of anæmia with leukopenia constitute positive differential criteria. Banti's disease, the terminal stage of splenic anæmia, is characterized by hypertrophic cirrhosis of the liver, jaundice, and ascites.

The diagnosis of leukæmia rests wholly upon the microscopical examination of the blood. Other conditions which, by reason of the presence of superficial lymphatic enlargements or splenic enlargement associated with more or less pronounced secondary anæmia, resemble leukæmia may be at once differentiated by the findings in the blood. It is only necessary in this connection to name the splenic tumor of chronic malarial infection; amyloid disease; malignant growths, cysts, and abscess; enlargements involving the left kidney, as hydronephrosis, cysts, perinephric cysts; abscess and malignant disease or cysts of the pancreas; and retroperitoneal sarcoma—all of which present resemblances to splenic tumor; and the hyperplasias of the lymphatic glands which occur in tuberculosis, syphilis, and malignant disease.

Hasty conclusions in a suspected case, in which the ratio of leucocytes to erythrocytes is normal, are unwarrantable because remarkable falls in the morbidly increased whites occur, (a) in the natural history of the disease; (b) during or immediately after acute intercurrent affections, as influenza, enteric fever, sepsis; (c) after the administration of drugs,—as arsenic and quinine,—the injection of nuclein, tuberculin, and antidiphtheritic serum, and the use of the X-rays.

Prognosis.—The outlook is in a high degree unfavorable. The number of permanent recoveries reported is limited. Remarkable and prolonged remissions occur. The influence of the X-rays upon the disease in some cases is to be considered. The acute lymphatic variety is peculiarly malignant and often runs a rapid course. The myelogenous form sometimes extends over eight or ten years, with remissions, even intermissions, and exacerbations. Progressive deterioration of the blood, hemorrhages, marked

gastro-intestinal disturbances (especially intractable diarrhœa), fever, dropsy, and massive enlargement of the spleen are of unfavorable significance.

Mikulicz's Disease.—There is gradual, symmetrical, painless enlargement of the lachrymal glands, followed by similar changes in the salivary glands. Special predisposing influences are not recognized, though males suffer more frequently than females and in the majority of the cases the disease has occurred between the twentieth and fortieth years of life. The condition is generally regarded as an infection, but a special pathogenic agent has not been demonstrated. It has been attributed to tuberculous infection, syphilis, and hypothyroidism. Howard, whose recent studies of the subject are of great importance, regards the cases as constituting not a simple morbid entity but a clinical syndrome varying in its etiology, form, and course, and comprising isolated and symmetrical disease of the lachrymal and salivary glands due to simple lymphomata, pseudoleukæmia, leukæmia, tuberculosis, and syphilis. This observer arranges the cases in three groups: (a) the simple form, in which only the lachrymal and salivary glands are involved, neither the adjacent nor distant lymphatics being affected and the blood picture remaining normal for years. (b) Pseudoleukæmia. In this group of cases the clinical manifestations are similar to the simple form except that the lymphatic glands, either locally or generally, are involved. The enlargement is variable in degree. Softening and caseation do not occur. The spleen may be enlarged. The blood may be normal or there may be a moderate secondary anæmia. In other cases there is a relative or absolute increase in the small lymphocytes, and in some cases large lymphocytes are present. (c) Leukæmia. In this group, in addition to the enlargement of the lachrymal and salivary glands of both sides there are the characteristic general enlargements of the lymphatic glands and a blood picture of leukæmia of the lymphatic type with the usual clinical phenomena—progressive weakness, irregular fever, slight œdema, and tendency to hemorrhage.

III. HODGKIN'S DISEASE.

Pseudoleukæmia; Infectious Granuloma.

Diagnosis.—A disease due to an infectious granulomatous involvement of the lymphadenoid tissue of the body and characterized clinically by enlargement of the lymph-nodes, marked anæmia, emaciation and a tendency to cachexia; anatomically by definite histological lesions in the affected glands and the presence of organisms of the diphtheroid type.

Etiology.—Predisposing Influences.—The disease usually appears in childhood, adolescence, or early adult life. It is very rare after the fourth decade. It is more common in males than in females in about the proportion of six to one. The occasional occurrence of cases in a parent or child, or among the children of the same family, about the same time, suggests rather the action of a local cause or direct infection than the hereditary or family transmission of the disease. Malaria, syphilis, and tuberculosis have been regarded upon wholly insufficient grounds as predisposing influences.

Exciting Cause.—The nearly constant presence of diptheroid organisms in the glands and their occasional presence in the blood, together with the results of experiments upon monkeys (Bunting, Yates) and the results obtained by the use of autogenous vaccines (Billings, Rosenow), point strongly to the infectious nature of the disease. Among the clinical facts which support this hypothesis are the following: preliminary or inflammatory irritation of the mucous membrane of the upper air-passages; disease of the tonsils, early localization in the cervical lymph-nodes, gradual extension from one group of lymphatic glands to another with recurrence of fever and the acute course of some of the cases.

There are clinical resemblances to lymphosarcoma, which are not borne out by the results of histological studies of the affected glands in the two diseases, and to glandular tuberculosis, which are negatived by the following facts: (a) absence of tubercle bacilli and the failure of inoculation experiments in a majority of cases studied; (b) absence of reaction to the tuberculin test in well-characterized cases; and (c) specific histological characters in Hodgkin's disease. The view that Hodgkin's disease is a form of lymphatic tuberculosis has arisen from the fact that secondary tuberculous infection not infrequently occurs, especially in the terminal stages.

Morbid Anatomy.—Histologically the presence in the lesions of fibroblasts, giant cells and eosinophiles is characteristic. The glands upon one side of the neck, very often the right, are usually first affected. The internal lymph-nodes are also generally enlarged—those of the thorax, the retroperitoneal, and the abdominal glands in the order named—and form large, firm masses, which give rise to the pressure symptoms which constitute striking clinical phenomena of the disease. The veins are especially liable to compression. The nerve-trunks and ureters do not always escape. The lymph-nodes even when greatly enlarged are not often adherent, nor is there a special tendency to capsular infiltration or invasion of contiguous structures. Caseation and necrosis do not occur in the absence of secondary infection. The spleen and liver are enlarged and the seat of scattered lymphoid masses.

Symptoms.—The most striking and usually the earliest clinical phenomenon is the enlargement of one or several lymph-nodes at the angle of the jaw. This enlargement is gradual and may attract little attention until several weeks have elapsed. The swollen glands are painless and may be recognized upon palpation as separate and distinct. They are not usually adherent to the skin. In some cases they are adherent among themselves. The peculiar adenopathy is progressive. The glands first involved gradually increase in size, adjacent groups in the lower part of the neck presently become implicated, then those in the axilla, the inguinal region, and sometimes those at the elbow and in the popliteal space. The lesions, at first unilateral, after a time appear upon the other side, but as corresponding groups are not usually enlarged to the same extent, they are irregularly symmetrical. The enlargement is, as a rule, most marked upon the side of the neck, where the masses often exceed the size of the fist and give rise to conspicuous deformities. It is not so great in the axillary and inguinal regions. The enlarged glands are at first of moderate

Fig. 333.—Hodgkin's disease.—Jefferson Hospital.

consistence and may be moved under the skin; later in the course of the disease they become harder and more or less adherent to the subjacent tissues. Tonsillitis, sometimes chronic, frequently precedes the early glandular changes in the neck; but this is not always the case and there are instances in which the lymph-nodes in this region remain unaffected in the presence of every other feature of the malady. The evidences of enlargement of the internal lymph-nodes do not usually show themselves until after the changes in the superficial groups have made considerable progress. In fact there are rare cases of Hodgkin's disease in which the external glands are not at all or but slightly increased in size. Lymph-nodes of the digestive tract, namely, the tonsils, the adenoid masses at the base of the tongue and in the pharynx, and the solitary and agminate glands of the intestines, are frequently implicated without giving rise to marked symptoms. The spleen is usually enlarged, but not to the extent often seen in chronic myeloid leukæmia.

Anæmia does not show itself until the malady has made some progress. After a time the changes in the blood, characteristic of anæmia of secondary type, develop rapidly in the acute cases; more gradually in the chronic forms of the disease. At a period when the symptom-complex is fully established, the condition of the blood is as follows: hæmoglobin decreased to about 50 per cent., not commonly much below this; color index usually low; moderate erythrocyte reaction, rarely below 2,000,000 per cubic centimetre; nucleated forms rare, when present of the normoblastic type; leucocytes normal or slightly increased, the increase often transient; relative increase of polynuclear neutrophiles or lymphocytes; no increase of eosinophiles.

In cases attended with pressure dyspnœa an erythrocytosis sometimes occurs with a count of 5,000,000 or more and a high percentage of hæmoglobin.

Three groups of symptoms may be recognized, namely those due to the enlarged glands, those due to the anæmia, and the cachexia. But the symptom-complex is not well defined.

(a) Symptoms Due to Lymphatic Hyperplasia.—*Superficial.*—External deformities and impairment of function, as of movement and the like. The prominence of these symptoms depends upon the location and degree of enlargement of the affected groups of glands. *Deep.*—Pressure symptoms, consisting of cough, dysphagia, dyspnœa, dilatation of superficial veins, cyanosis, local disturbances of circulation, various dropsies, œdema, and effusions into the serous sacs. Derangements of function due to pressure doubtless contribute to the cachexia. Neuralgias in various distributions are caused by pressure upon deep or superficial nerve-trunks.

(b) Symptoms Due to the Anæmia.—Pallor, headache, dyspnœa in some cases, asthenia, anasarca, hemorrhages, and fever. The last symptom may occur as a mild, irregular pyrexia early in the course of the disease, the attacks often being associated with sudden increase in the size of the affected glands; as an irregular ague-like paroxysmal fever when the disease has reached an advanced stage; and finally in the form of the "chronic relapsing fever" of Pel, or "recurring glandular fever" of

Ebstein, in which febrile periods of from ten to fourteen days' duration, and either intermittent or remittent in type, alternate with irregular periods of apyrexia lasting several days. In view of the frequency with which intercurrent and terminal infections occur in Hodgkin's disease, the likelihood that these forms of fever are not primarily symptomatic of the disease under consideration must be considered.

(c) The Cachexia.—The general disturbances of nutrition ultimately reach a high grade. Extreme pallor, emaciation, profound asthenia, bronzing of the skin in some of the cases, hemorrhages into the skin and from various mucous surfaces, together with the tumor masses in the inferior carotid triangle and the occipital region and elsewhere, constitute a striking and most suggestive clinical picture.

Troublesome pruritus and other cutaneous manifestations are early symptoms in many cases and patients complain of loss of appetite, fatigue, shortness of breath and cardiac palpitation on exertion and a falling off in mental power.

Diagnosis.—The direct diagnosis rests upon the association of a widespread affection of the lymph-nodes involving progressively superficial and deep chains, anæmia of high grade and secondary type, and cachexia. The researches of Reed, Longcope, and others have settled the question as to the specific nature of the histological lesions in Hodgkin's disease and the uncertainties as to its relationship with tuberculosis, lymphosarcoma, and leukæmia. The association of the following changes is characteristic: proliferation of connective-tissue stroma resulting in fibrosis, and in a majority of the cases numbers of eosinophiles. These facts fully justify the excision of one or more superficial glands in a doubtful case for histological examination.

Differential.—*Tuberculous adenitis* rarely develops after the twenty-fifth year. The enlargement frequently involves the glands of the neck upon one or both sides. Inflammatory adhesions between the individual glands and to the adjacent structures, and in particular to the overlying skin, are the rule. There is tenderness, and a tendency to softening, suppuration, and caseation with sinus formation. The process is sluggish and mostly limited to one or two groups of glands, rarely extending to distant regions. The signs of tuberculous lesions in the mouth (especially in the tonsils), in the skin, bones, lungs, or other viscera are suggestive. The presence of tubercle bacilli in the excised glandular tissue is suggestive of a tuberculous process, but the coexistence of the two diseases is by no means rare, while the definite histological lesions of Hodgkin's disease are distinctive. In afebrile cases the tuberculin tests may be employed.

Syphilitic Adenitis.—The glands in direct relation to the initial lesion, usually the inguinal, are commonly first affected, the glands of the neck being primarily involved only when there are buccal or labial lesions. The adenopathy is general, with an especial tendency to implication of the postcervical and epitrochlear glands. The enlargement is moderate, firm, painless, and not accompanied by a tendency to form adhesions to contiguous structures. A history of exposure, the presence or vestigia of an initial lesion, secondary manifestations, and the effects of mercurial treatment are of diagnostic importance. The Wassermann test may be tried.

Leukæmia (see p. 552).

Lymphosarcoma.—The conditions are very often mistaken for each other. In lymphosarcoma the masses are of rapid growth and may attain great size; adhesions among individual glands and to adjacent structures, with inflammatory changes attended by redness, tenderness, and local œdema, are common. Softening and fluctuation suggestive of abscess formation are frequent. The cutaneous veins are often enlarged and tortuous; pain is sometimes a prominent symptom, and there are progressive infiltrations and metastases.

It is important in the differential diagnosis of Hodgkin's disease that this affection is characterized by a progressive implication of successive groups of glands, while in those affections, with the exception of syphilis, to which it may sometimes bear a superficial resemblance, the lesions are limited to single glands or, at most, to two or three groups which are anatomically related and become affected at or about the same time.

Prognosis.—The disease runs its course with exacerbations of varying intensity and remissions of irregular duration. Acute cases may end fatally in two or three months. Very chronic forms may last three or four years. The average duration is about one year. Urgent pressure symptoms, intense anæmia, hemorrhages, and early cachexia are of unfavorable prognostic significance. The outcome is lethal, but remarkable improvement in the blood and other clinical phenomena has followed the use of the X-rays, the early extirpation of the enlarged glands, and the administration by the mouth and subcutaneously of arsenic in the form of Fowler's solution, or of sodium cacodylate. Recently autogenous vaccines have been followed by improvement.

IV. DISEASES CHARACTERIZED BY HEMORRHAGE.

The Hemorrhagic Diathesis; Morbus Maculosus.

The essential symptom is hemorrhage, which may be into the skin, from the mucous surfaces, subserous, or into the viscera.

(a) Purpura.

Definition.—An affection which appears spontaneously, and is characterized by hemorrhages into the skin, mucous membranes, and internal organs. The following forms are recognized: (1) purpura simplex; (2) purpura hæmorrhagica or morbus maculosus Werlhofii; (3) purpura rheumatica or peliosis rheumatica; (4) purpura with visceral symptoms; and (5) symptomatic purpura. There are those, as Litten, who regard these as manifestations of the same disease, differing in intensity.

Purpura always occurs as a sporadic affection and never, like scurvy, in local outbreaks or as an endemic disease, or like the hemorrhagic cases of the infections—variola, varicella—during the course of epidemics. It is not a common disease.

Etiology.—Predisposing Influences.—Females are somewhat more disposed than males. The greatest liability is in middle life, but neither infancy nor old age escapes. Purpura sometimes has developed during the convalescence from enteric fever, malaria, scarlet fever, and measles,

and in the course of all forms of nephritis, especially the interstitial variety. Intense nervous shock, such as attends fright, fever, or pain, has been followed by the occurrence of purpura.

Exciting Cause.—No immediate cause can usually be discovered. The hypothesis that purpura is an infectious disease has not yet received demonstrable support. Unfavorable conditions of life, damp and insalubrious dwellings, and chilling of the body have been regarded upon inadequate grounds as etiological factors.

The etiological factors in the hemorrhagic diseases remain obscure, chiefly owing to the uncertainty concerning the physiology of the coagulation of blood and the spontaneous cessation of bleeding from open vessels—attempts to classify these affections according to the part played in departures from the normal coagulability in fibrinogen, calcium, prothrombin, antithrombin, blood platelets and fibrin-dissolving ferments have not been successful.

1. **Purpura Simplex.—Symptoms.**—Isolated hemorrhagic spots upon the skin may constitute the only manifestation of the disease. They vary from a mere point in diameter to patches of considerable size. The latter are not common in simple purpura. Purpuric spots are more commonly and more abundantly distributed on the lower extremities than elsewhere. They are at first bright red in color, but rapidly become darker, and fade, leaving patches of brownish pigmentation which gradually undergo resorption. These spots do not disappear upon pressure or when the skin is made tense. In some cases the spots appear abruptly and wholly without prodromes, more commonly they are preceded by symptoms such as attend the initial stage of a mild infection, lassitude, loss of appetite, nausea, and fever. The disease sometimes lasts but a day or so and is marked by a single crop of hemorrhages into the skin; in other cases its course may extend over a week or two and be marked by successive crops of petechiæ. The spots are scattered over the arms, abdomen, and legs. They are rarely seen upon the face. The course of this variety of purpura is favorable. In a few days, or at most a fortnight, the spots have disappeared and the patient has regained his usual health. A slight degree of anæmia and transient albuminuria may occur during convalescence. It is much more common in children than adults, and is often associated with diarrhœa and slight rheumatoid pains and swelling of the joints.

Diagnosis.—The direct diagnosis of purpura simplex may be made when, in the absence of a primary antecedent affection, the purpuric eruption appears after trifling derangements of health and disappears in the course of a few days or at most a week or two. The differential diagnosis from other forms of purpura depends upon the mildness of the attack, its short duration, and the absence of special characteristic manifestations, such as copious hemorrhages into the skin, hemorrhages from mucous surfaces, fever, joint affection, urgent gastro-intestinal symptoms, tendency to recur, and so forth.

2. **Purpura Hæmorrhagica; Morbus Maculosus; Werlhof's Disease.**—The cutaneous hemorrhages are abundant and extensive, and there is bleeding from mucous surfaces. This form of purpura sometimes attacks individuals in apparent health, but is far more common in delicate children

or adolescents, especially girls. It may also occur without prodromes and run an afebrile course. When fever is present it is of moderate intensity—101°–102° F. (38.5°–39° C.). Gastric disturbances, a mild rheumatoid affection of the joints, and albuminuria occur in many of the cases. The cutaneous hemorrhages are profuse and extensive, and in some instances extensive areas or even the greater part of the surface of the body are involved, so that the discolored space in some portions of the body greatly exceeds that of the normal skin. Frequently, but not always, there is bleeding from mucous surfaces, usually first in the form of epistaxis, which is soon followed by hemorrhage from the buccal and gingival mucous surfaces, hæmaturia, hæmoptysis, and bleeding from the bowel. Under these circumstances profound anæmia may rapidly develop. Death may result from the loss of blood. PURPURA FULMINANS.—A malignant variety which occasionally occurs in children may cause death in the course of twenty-four or forty-eight hours from extensive cutaneous hemorrhage with or without bleeding from mucous surfaces. The course of the disease is often protracted, with frequent relapses, which are apt to occur when the patient attempts to leave his bed, and leads to great debility and anæmia, with vertigo, cardiac palpitation, and syncopal attacks. The DIRECT DIAGNOSIS of Werlhof's disease depends upon the severity of the affection, the prominence and extent of the cutaneous hemorrhage and bleeding from mucous surfaces.

It is to be differentiated from purpura simplex on the one hand by the above criteria, and from purpura rheumatica upon the other by the infrequency and mildness of the arthritis, the absence of urticaria and erythema, the tendency to hemorrhage from mucous surfaces. From scurvy it is to be distinguished by the conditions under which the disease develops, its more or less abrupt occurrence in an individual of fair previous health, and the absence of swelling of the gums; from the malignant or hemorrhagic forms of the infectious diseases by the symptoms of the onset in the latter, the gravity of the attack, the first appearance of petechiæ upon the forehead and wrists, their association with abortive eruptions or extensive suggillations.

3. **Purpura Rheumatica; Peliosis Rheumatica; Schönlein's Disease.**—This hemorrhagic affection is characterized by an eruption in which the spots "never coalesce," a multiple arthritis, and protracted course. It occurs in young adults, chiefly in males and usually in individuals who have a delicate, highly vascular skin and have previously suffered from rheumatism. It frequently begins with angina tonsillaris, fever of moderate intensity,—102° F. (39° C.),—and rheumatoid pains in the joints, especially the ankles, knees, the joints of the hands, and the shoulders. The affected joints are painful to the touch and upon movement and the seat of endo- and periarticular exudate. The eruption appears first upon the lower extremities or over the affected joints. It is frequently merely purpuric, but may show urticarious wheals, erythematous patches, or infiltrated areas suggestive of erythema nodosum. Vesication may occur. Local œdema occasionally appears, especially about the face—*febrile purpuric œdema*. The eruption tends to recur in crops and may continue to reappear for several weeks. The patients are sensitive to cold, and

fresh outbreaks occur upon rising from bed or any chilling of the surface. Attacks at the same season in successive years have been noted. The urine has no special characters. It sometimes contains albumin. The DIRECT DIAGNOSIS of Schönlein's disease rests upon the association of a well-defined joint affection with purpura, which tends to recur in crops, erythema, and local œdema. The joint affection is usually mild and shows neither the evanescence characteristic of rheumatic fever nor the persistence of gonorrhœal arthritis. DIFFERENTIAL.—The existence of Schönlein's disease as a nosological entity has been much questioned. It has been regarded as true rheumatism, from which it is easily distinguished by the eruption, which tends to recur, and the almost constant absence of cardiac lesions; as a form of gonorrhœal arthritis, a view that cannot be entertained, since gonorrhœa, though sometimes present, is mostly absent; as an intense form of purpura with rheumatoid symptoms, an opinion which finds some support in the fact that mild multiple arthritis is not uncommon in the hemorrhagic diseases.

4. **Purpura and Visceral Symptoms; Purpura Abdominalis; Henoch's Purpura.**—The cutaneous hemorrhages are accompanied by abdominal symptoms, vomiting, diarrhœa, often hemorrhagic, and colic. These symptoms occur in paroxysms separated by intervals of several weeks or months. The disease is most common in childhood and adolescence, but may occur in early adult life. It is much more common in males than females. It frequently attacks individuals living in poverty and want, but is by no means unknown among the affluent. There is a varying period of impaired health with headache, weakness, and loss of appetite, followed by subacute or acute arthritis affecting one or more joints, and moderate fever. Purpura now appears, often but not invariably upon the legs and feet, but spreading to the trunks and elsewhere, and being particularly abundant in the neighborhood of the affected joints. The hemorrhagic eruption is associated in varying degree with erythema, patches of œdema, and urticaria. Coincidently with the eruption the above mentioned abdominal symptoms appear. Epistaxis and hemorrhage from other mucous tracts occur. The colic, most intense about the umbilicus, is often especially severe and protracted; the abdomen is retracted and tender; there is complete loss of appetite; retching is frequent and distressing; the spleen is enlarged; the pulse small and frequent, and in many instances there is alarming exhaustion. In fact it is to this variety of purpura that the rapidly fatal *purpura fulminans* is to be referred. Nephritis is of frequent occurrence and may persist. These symptoms in the ordinary cases undergo gradual amelioration, and the infant enters upon an apparent convalescence, only to suffer from similar paroxysms at varying intervals. The duration is variable. The attack may last for several days, and recurrences may extend over a period of months or even years. The prognosis in children is favorable—mortality less than 5 per cent.; in adults less so—mortality about 23 per cent.

The DIRECT DIAGNOSIS of this form of purpura rests upon the association of purpura with erythema and urticaria, visceral symptoms, especially the abdominal crises, and nephritis, the joint affection and fever, and the paroxysmal nature of the recurrent attacks. This affection, con-

cerning the cause of which we have no definite knowledge, presents a well-defined symptom-complex. The differential diagnosis requires no special consideration.

The blood changes in the various forms of purpura are similar and without diagnostic significance. They are those of secondary anæmia of varying intensity, with leucocytosis. The coagulation time is much protracted, in some cases reaching ten or even fifteen minutes.

5. **Symptomatic Purpura.**—Purpura is a symptom, not a disease, yet in the foregoing affections it is a symptom of such prominence as to justify the older use of the term to designate a group of diseases the etiology of which is as yet unknown, and of which cutaneous hemorrhage is the only common and constant phenomenon. But purpura is also a very common symptom in many conditions and well-defined diseases. This form is known as symptomatic purpura. Traumatic.—The ecchymosis which follows a blow or contusion does not differ in appearance or course from similar cutaneous hemorrhages occurring in disease. Mechanical purpura may result from severe transient venous stasis, as in whooping-cough or epilepsy, or more prolonged interference with the circulation, as the pressure of a splint or bandage. Toxic.—The venom of snakes causes rapid and extensive blood extravasations. Jaundice, especially when prolonged, is frequently associated with petechiæ. Among the drug exanthems purpura occupies an important place and may follow the administration of copaiba, ergot, quinine, belladonna, mercury, and the iodides. There is usually an idiosyncrasy. The question as to whether the cutaneous hemorrhage is due to the drug or the disease for which the drug has been given is always to be considered. There is no question as to the significance of a petechial rash in ergotism or the specific effect of the iodides in causing this symptom in certain cases. In the case of the iodides, coryza, angina tonsillaris, erythema, and fever may accompany the purpura. These symptoms may quickly follow small doses of the drug. Infectious.—The ordinary rashes of typhus and cerebrospinal fever are purpuric; similar lesions of the skin occur in sepsis and especially in malignant endocarditis; petechiæ and ecchymoses characterize the malignant forms of measles, scarlet fever, and smallpox. In that form of variola known as purpura variolosa there are petechiæ and most extensive suggillations of blood. Cachectic.—The terminal dyscrasia of cancer, chronic nephritis, Hodgkin's disease, tuberculosis, and other incurable wasting diseases is frequently accompanied by petechial eruptions, usually confined to the lower extremities. A similar form occurs in old age and prolonged want of food, and extensive purpura is characteristic of scurvy. Neurotic.—Myelopathic purpura occasionally occurs in spinal diseases, particularly forms of myelitis, especially transverse myelitis. The bleeding may be associated with trophic disturbances, erythema, and localized sweating. It occurs also in rare instances in tabes in the course of the lightning pains, and in association with herpes, œdema, and local sweating. Purpura may occur in the distribution of the affected nerve in severe neuralgia. The "stigmata" or bleeding points of hysteria, when not artificially produced for purposes of deception, must be referred to this category.

(b) **Hæmophilia.**

Bleeders' Disease.

Definition. — A constitutional anomaly mostly hereditary, — *vitium primæ formationis*,—very rarely acquired, which manifests itself by the occurrence of uncontrollable bleeding, either spontaneous or from traumatism which may be slight, and occasionally by hæmarthrosis sometimes followed by permanent deformity.

Hæmophilia is a hereditary and congenital condition; hemorrhage, which shows neither tendency to stop nor yields to treatment, the sign by which it makes itself known. In the absence of a history and of bleeding, there are no indications of the constitutional fault.

Etiology. — Predisposing Influences. — Grandidier has described hæmophilia as the most hereditary of all hereditary diseases. The occasional occurrence of fatal hemorrhage from trifling wounds has long been known, but the transmission of the tendency from generation to generation has been especially studied during the last century. In the well-known Appleton-Swain family there have been bleeders for nearly two hundred years. Grandidier gives the history of 200 bleeder families. In some instances the transmission is direct from the parents to the children. Usually, however, the transmission follows a peculiar law of heredity, namely, that the females transmit while the males acquire the condition. Exceptionally females also acquire it. From this it follows that hæmophilia is much more common in males than in females. The actual ratio is about 13 to 1. It is the rule that in a bleeder family a woman not a bleeder transmits the condition to her children without having acquired it herself, a generation having thus escaped. Men who are bleeders, but whose wives are not descendants of bleeder families, do not always beget bleeder children, and men who are members of bleeder families, but themselves not bleeders and whose wives do not belong to bleeder families, rarely beget bleeder children. Certain lines in bleeder families thus tend to become normal. It has occasionally happened that children born to parents neither of whom belong to bleeder families have been bleeders—*congenital hæmophilia, spontaneous hæmophilia.* Hæmophilia is more common in Germany, England, and the United States than elsewhere. The condition is usually discovered in infancy or early childhood. Social conditions are apparently without influence. In some bleeder families a neuropathic constitution has been recognized. As a rule the stock is fine, the families large, the members healthy and robust looking, with good skins and delicate complexions.

The actual cause of hæmophilia remains unknown

Pathogenesis. — Various theories have been advanced. Among them are habitual disproportion between the volume of blood and the vessels; hydræmic plethora; abnormal composition of the blood; an increase in the red corpuscles—erythrocythæmia; fragility of the vessels; pathogenic infection; and deficiency in the fibrin ferment. None of these has met the requirements of the condition.

Symptoms.—The existence of hæmophilia is usually discovered in consequence of trauma. The hemorrhage cannot be controlled, or is

arrested with difficulty and only after prolonged effort. There are various grades of severity, from the mildest to the most severe which terminates fatally. The hemorrhage may be spontaneous or traumatic. The traumatism is often so slight as to escape attention, as the contusion resulting from a slight fall or blow, or chastisement. Spontaneous hemorrhages are sometimes preceded by fulness in the head, vertigo, tinnitus, palpitation, and nausea. They may be superficial, as from mucous surfaces, namely, that of the nose, mouth, female genitalia, urinary passages; or from the lungs, stomach, or intestines, or finally from cicatrices or ulcers upon the skin. Interstitial spontaneous hemorrhages are usually superficial. They chiefly occur in the scalp, face, scrotum; less frequently upon the extremities, and rarely upon the trunk. It is probable that the majority of such cases are in point of fact the result of slight traumatism. They consist of petechiæ, ecchymoses, and subcutaneous hæmatomata. The ordinary forms of trauma by which hæmophilia is manifest in external hemorrhage comprise abrasions, scratches, cuts, wounds, and surgical incisions. The bleedings named in the order of frequency are from the nose, mouth, bowels, urethra, vulva, stomach, lungs. Less frequently continuous bleeding takes place from areas of skin, especially upon the head and the scrotum, the tongue, eyelids, conjunctiva, finger-tips, lobe of the ear, and vulva. Bleeding from the head is far more common and usually more severe than from the extremities or trunk. Trifling operations, as lancing the gums, the extraction of a tooth, circumcision, or venesection, have been followed by fatal hemorrhage. Lethal hemorrhage has followed the rupture of the hymen in coitus. The bleeding usually is of the type described as parenchymatous; there is capillary oozing, more or less abundant, from every point of the exposed surface. It is not common to find flowing vessels of any size. It is continuous and may last for hours or for many days. After a time syncope may occur and the bleeding cease. Prolonged bleeding is often followed by death. Epistaxis may prove fatal in twenty-four or thirty-six hours. The coagulation time of the blood is much retarded. With Wright's instrument it has varied from twenty to forty-five minutes as compared with three to six minutes with normal blood. The arthropathies of hæmophilia are rheumatoid in character. They occur both spontaneously and after contusions. The knees and elbows are most commonly affected. The onset is acute, with swelling, pain, redness, and slight fever. Less commonly there is hæmarthrosis without fever. Repeated hemorrhage into the joints and muscles may occur in the absence of external or subcutaneous bleeding, and give rise to a false diagnosis of chronic rheumatism or tuberculosis. The resulting deformities sometimes suggest arthritis deformans.

Diagnosis.—Direct.—The recognition of hæmophilia depends upon the family history and the occurrence of persistent or uncontrollable hemorrhage. Neither the family history alone, since there are members of bleeder families who are not bleeders, nor a single unmanageable bleeding from a trifling cause, since difficult local hemorrhages are common enough in those who are not hæmophilic, justifies a positive diagnosis. The association of an hereditary tendency to repeated stubborn bleeding from slight injury with arthritis is highly suggestive. Even heredity is absent

in the spontaneous or congenital cases. DIFFERENTIAL.—Habitual epistaxis and hæmaturia are not attended with the tendency to hemorrhages from slight wounds or cuts. In the hereditary local bleedings from the nose or mouth, associated with telangiectasis of mucous membranes, and nævi, the blood losses arise from definite lesions, and arthropathies are absent. Purpura rheumatica presents points of resemblance to hæmophilia, especially in the prominence of the arthritis. There may be more than one case in a family, but the peculiar form of heredity seen in bleeder families does not occur, and there are multiple spontaneous hemorrhages rather than excessive bleedings from limited surfaces.

Prognosis.—The outlook is unfavorable; many of the cases die in early infancy, a majority before puberty. The hæmophilic tendency becomes less marked as life advances, but the subjects rarely reach seventy. The prognosis is less favorable in boys than girls. Death does not often occur in a first bleeding, but it occasionally results from uncontrollable bleeding after ritual circumcision. Females who are hæmophilic are apt to menstruate early and freely, but neither this function nor that of parturition is attended in bleeder families with an especial tendency to dangerous blood loss. Any form of hemorrhage may, however, prove fatal; that which is most frequently so being epistaxis.

(c) The Hemorrhagic Diseases of the New-born.

Acute Fatty Degeneration of the New-born; Buhl's Disease.—This rare affection is characterized by fatty degeneration of the heart, liver, and kidneys, and hemorrhages in the various organs. The chief symptoms are inanition and external hemorrhages, of which the more common are omphalorrhagia, melæna, and hæmatemesis. Bleeding may also take place from the mouth, nose, eye, and ear. The infant is born in a condition of asphyxia, from which resuscitation is only partial, and dies at once or in the course of a week or ten days. The skin is cyanotic and icteric. An anatomical diagnosis cannot be made in the absence of a microscopical diagnosis. It is therefore probable that the condition is very often overlooked. The differentiation from phosphorus and arsenic poisoning, in which similar parenchymatous changes in the viscera occur, may be apparent from a study of the circumstances; from sepsis, with interstitial hemorrhages and fatty degeneration, it may be difficult. Infection by way of the cord must be excluded. The prognosis is lethal.

Infectious Hæmoglobinuria of the New-born; Epidemic Hæmoglobinuria; Winckel's Disease.—This obscure affection arises as an endemic or epidemic disease in lying-in hospitals. It begins about the fourth day of life and is characterized by marked cyanosis with icterus, hæmoglobinuria, somnolence, and collapse without fever. Vomiting and diarrhœa are common. The urine contains small amounts of albumin and methæmoglobin. Epithelial granular and blood-casts are also present. It may attack strong, well-developed children. It runs a rapid course and is extremely fatal. Death may be preceded by convulsions. The etiology is unknown. The post-mortem findings are in some cases similar to those of Buhl's disease. The spleen is enlarged. The diagnosis rests upon the occurrence of asso-

ciated cyanosis and icterus, the sudden onset upon the fourth day, the character of the urine, and the endemic or epidemic prevalence of the affection in an institution. The disease is to be distinguished from Buhl's disease by its onset some days after birth and the urinary conditions, and from icterus neonatorum by the severity of the process.

"Hemorrhagic Disease of the New-born"; Morbus Maculosus Neonatorum. — Townsend has made a thorough study of a condition of not infrequent occurrence and uniform symptomatology, which has been described under the above terms. This affection is self-limited, attended with moderate fever, and occurs almost always within the first week of life. Hemorrhage arises from the mouth, nose, bowels, and navel. Petechiæ and ecchymoses are common. Visceral hemorrhages and bloody collections in the serous sacs are found upon post-mortem examination. The children are very anæmic in appearance, but the peripheral blood may show an increase in hæmoglobin and erythrocytes. In a case of Townsend's the hæmoglobin was 125 per cent. and the erythrocytes 6,245,600. The affection is of brief duration. The mortality is about 60 per cent. When recovery takes place it is usually complete and permanent. The diagnosis may be based upon the general character of the disease, its manifestly infectious nature, its self-limited course, and its prevalence in institutions. It is to be distinguished from other forms of disease of the new-born, characterized by hemorrhage, which have been described, and especially from hæmophilia. A general rather than a local tendency to hemorrhage is of diagnostic significance. It is probable that many of the cases described as melæna neonatorum belong to this category.

V. DISEASES OF THE SPLEEN.

i. Anatomical Anomalies.

Complete absence of the spleen occasionally occurs in association with other developmental faults. Much more commonly the organ is rudimentary. Very frequently there are accessory spleens — *splenunculi* — the supernumerary spleen or spleens lying within the folds of the gastrosplenic omentum and other processes of the peritoneum passing to the spleen. Abnormal lobulation and departures from the usual shape are common. These abnormalities are of no clinical interest. Entire absence of the spleen may be unattended by functional disturbances.

In complete *transpositio viscerum* the spleen occupies a position upon the right side corresponding to its normal position on the left. Under these circumstances it is sometimes represented by a number of lienculi which may be arranged in a cluster or loosely separated. Exceptionally the transposition may involve only the liver and spleen. Displacement may occur in the new-born downward as the result of abdominal deformities or umbilical hernia, upward in consequence of congenital diaphragmatic hernia. These displacements are of minor clinical interest.

ii. Movable Spleen.

Lien Mobilis; Wandering Spleen.

This condition is sometimes the result of congenital elongation of the gastrolienal ligament; sometimes of a similar elongation acquired through mechanical influences, such as pressure, blows, the succussion of violent, continuous coughing, the traction of peritoneal adhesions, and the weight of the enlarged organ itself. Wandering spleen is most commonly encountered in women suffering from enteroptosis.

Symptoms. — The dislocation is downward and forward, and the organ may reach a position below the level of the umbilicus or even pass into the pelvis, or it may form part of the contents of a large inguinal hernial sac. It is, as a rule, more or less enlarged. Subjective symptoms may be wholly absent, and the condition may be accidentally discovered. More commonly there are sensations of weight and dragging, with diffuse dull pain in the left flank. Colic, constipation, dysuria, and neuralgia may result from derangement of the various structures upon which the displaced and enlarged spleen exerts traction or pressure. The obstruction to the circulation sometimes causes great distention of the splenic vein. Torsion of the pedicle may lead to strangulation with great pain, tenderness, and local swelling, followed by necrosis with local or general peritonitis.

Physical Signs.—The upper end of the organ may sometimes be felt below the edge of the ribs, an important diagnostic point in the differentiation between an enlarged and a dislocated spleen. Palpation detects the indented median, the outer rounded, and the sharp lower border. The respiratory movements do not affect the dislocated spleen as they do the normal or merely enlarged organ. The normal dulness in the left hypochondrium is replaced by tympany.

Diagnosis.—The diagnosis is usually unattended by difficulty. The size, shape, and position of the organ, its free mobility, and the absence of dulness in the normal position of the spleen serve to distinguish it from the various abdominal or pelvic tumors, which its presence in an abnormal position might suggest.

iii. Acute Splenic Tumor.

The spleen undergoes enlargement in the acute febrile infections. The degree of enlargement varies in different diseases and in different cases of the same disease. The enlargement is almost constant in malaria, and so common in enteric fever as to constitute a phenomenon of diagnostic importance. It is also very frequent in typhus and relapsing fever, and occurs in pneumonia, smallpox and the other exanthemata, anthrax and septic conditions. Moderate splenic enlargement is frequently observed in acute miliary tuberculosis, secondary syphilis, and cerebrospinal fever. It occurs also, but is less common and less marked, in various acute catarrhal inflammatory conditions of the respiratory system, as coryza, tonsillitis, and bronchitis.

Symptoms are usually wholly absent. In exceptional cases there are sensations of weight and tension in the left flank, and some discomfort is experienced when pressure is made over the splenic region.

Physical Signs.—Inspection.—In rare instances in persons with little subcutaneous fat the side may slightly bulge and the outline of the lower border of the enlarged viscus may be seen below the margin of the ribs, especially upon deep inspiration. Palpation.—By this method moderate degrees of splenic enlargement may be recognized. The patient should be partly upon the right side, with his knees and thighs moderately flexed, and his head and shoulders supported upon a pillow. The physician standing to the left of the patient performs bimanual palpation, the palms of his right hand exerting pressure over the splenic area in the posterolateral aspect of the chest, while the fingers of his left hand are passed firmly upward beneath the margin of the ribs in front, the patient at the same time being directed to breathe slowly and deeply with an open mouth. The spleen descends with each deep inspiration and if enlarged may be readily felt by the fingers of the left hand. When the enlargement is considerable, the notches in the anterior border may be palpated, and in very rare instances pulsation has been recognized. Percussion.—This method of examination yields unsatisfactory results in moderate degrees of enlargement and cannot be depended upon. Errors occur in gaseous distention of the stomach, meteorism, fecal accumulations in the colon, and enlargement of the left kidney. When the spleen is considerably enlarged this method is more satisfactory. Very light direct percussion with the dorsal surface of the finger-tip yields satisfactory results, as does auscultatory percussion. *Auscultation.*—Intermittent and continuous soft murmurs have been heard in the splenic area in the malarial paroxysm and in relapsing fever.

iv. Chronic Splenic Tumor.

Hypertrophy of the Spleen; Congestive Hypertrophy.

Chronic enlargement may follow acute splenic tumor and be due to the action of infectious principles. It occurs also in chronic malaria, leukæmia, cirrhosis of the liver, and cardiac affections—*stasis spleen.* The organ may be increased to twenty-five or thirty times its normal size. Its surface is commonly smooth and there is thickening of the capsule.

Symptoms. — When the enlargement is of moderate size there are often no subjective symptoms. When it is considerable, weight, dragging, and a dull pain in the left side are experienced. Interference with the respiratory play of the diaphragm may cause dyspnœa, especially when the patient lies upon his left side. Traction upon the stomach may lead to loss of appetite, indigestion, nausea, and vomiting. Cardiac palpitation, œdema of the ankles, and colic occur. Anæmia is often marked. Hemorrhages and especially hæmoptysis are occasionally present.

Physical Signs.—The left side of the abdomen and the left hypochondrium may be distended. The visible and palpable tumor is popularly known in malarial districts as "ague cake." The lower and anterior mar-

gins are often distinctly palpable with their characteristic features, and may, in extreme cases, reach to the brim of the pelvis and to the right of the median line respectively. Upon auscultation friction sounds may sometimes he heard. The bruits occasionally heard in acute splenic tumor are not present. In cases of moderate enlargement in which there are no adhesions, the spleen may be so freely movable as to constitute one of the forms of movable spleen.

Diagnosis.—Direct.—The physical signs elicited upon inspection, palpation, and percussion, the presence of dulness in the normal splenic area, together with its uninterrupted extension to abnormal limits downward and forward and the well-defined borders notched in the anterior and rounded in the inferior lines, and more or less distinct participation of the tumor mass in deep respiratory movements of the diaphragm justify a diagnosis of hypertrophy of the spleen.

Differential. — The nature of the enlargement may be obscure. In leukæmia the condition of the blood is diagnostic. In Hodgkin's disease the history is important, and the presence of enlarged superficial lymph-nodes, especially when they form groups or masses in the cervical, axillary, or inguinal regions, is suggestive. In congenital syphilis and rickets the associated phenomena are significant. In splenic abscess, fluctuation and fever, together with other septic phenomena, point to the presence of pus. Echinococcus cysts of the spleen are rare and may give rise to uncertainty. Rupture into the intestine or externally may afford clinical evidences by which the true condition may be recognized during life, and rupture into the peritoneum may cause fulminant peritonitis, but these accidents are among the most infrequent of clinical occurrences. Malignant disease of the spleen is attended with grave disturbances of the general health, cachexia, and metastasis.

Tumors of the kidney usually occupy a lower position and are less movable. They are crossed diagonally by the colon and often associated with urinary symptoms of importance. Nephromata are common in early life, and hydronephrosis and pyonephrosis at all ages. A diagnosis of splenic hypertrophy or abscess should never be made until after every form of renal tumor has been excluded. A tumor of the fundus of the stomach, of the colon, or the omentum may, as a rule, be differentiated without difficulty from enlargement of the spleen.

Moderate enlargement is usually present in movable spleen. The latter condition is characterized by the free movement of the organ, its contour, and by resonance in the region of normal splenic dulness.

Prognosis.—The outlook depends upon the cause of the enlargement. Congestive and malarial spleens often undergo remarkable diminution in size. The leukæmic spleen shows wide oscillations in volume in the course of the disease and under treatment. Enlarged spleens may become smaller during pregnancy. The enlargement may exist for years without detriment to health.

v. Splenic Tumor with Anæmia.

Primary Splenomegaly; Splenic Anæmia; Banti's Disease.

Anæmia is characteristic of many conditions in which the spleen is enlarged, especially the primary anæmias, as leukæmia and pernicious anæmia. Secondary anæmia accompanies splenic enlargement in Hodgkin's disease, chronic malaria, and various forms of hepatic cirrhosis. Idiopathic splenomegaly with secondary anæmia occurs in a group of cases of which the following types are the most important:

(a) PRIMARY SPLENOMEGALY.—Marked and persistent enlargement may occur without associated disease and with but slight blood changes, and give rise to no symptoms other than those caused by the pressure and weight of the enlarged organ.

(b) SPLENIC ANÆMIA.—The spleen is very large. There is marked anæmia of secondary type. Hemorrhages, particularly hæmatemesis, are common. Purpura, melanoderma, and œdema of the lower extremities occur. The disease runs a very chronic course.

(c) BANTI'S DISEASE.—**Definition.**—Primary splenomegaly with anæmia and secondary cirrhosis of the liver.

Etiology.—Banti's disease occurs in adolescence or early middle age. The causal factors are unknown.

Symptoms.—The disease may be divided into three stages. *First Stage.*—There is progressive enlargement of the spleen with anæmia. In some cases the liver is slightly enlarged and there is faint but inconstant jaundice. The blood-picture shows a decreased number of red corpuscles. The color index is low. There is no constant change in the white corpuscles: in some cases, however, there is a leukopenia with relative increase in the lymphocytes. This stage is of long duration.

Second Stage.—Gastro-intestinal symptoms are troublesome and the jaundice deepens. The anæmia becomes more intense and cachexia develops.

Third Stage.—The signs of hepatic cirrhosis are now well advanced. Ascites is common: the cachexia becomes more pronounced and death from œsophageal hemorrhage may occur.

Prognosis.—The early recognition of Banti's disease is of the highest importance. Splenectomy in the first stage is frequently followed by a complete restoration to health. When the case has reached the second stage the operation fails to arrest further progress of the disease. The differentiation from myeloid leukæmia is necessary since the removal of the spleen in the latter affection is almost always followed by death.

Diagnosis.—The DIRECT DIAGNOSIS of splenic anæmia rests upon the association of primary splenomegaly with secondary anæmia, and the absence of enlargement of the lymph-nodes.

The DIFFERENTIAL DIAGNOSIS between splenic anæmia and pernicious anæmia depends upon the morphological characters of the blood, the relatively high hæmoglobin percentage, and small size of the spleen in the latter affection. Leukæmia may be at once differentiated by the blood picture, and Hodgkin's disease by the enlargement of the lymph-glands and their peculiar massing in the cervical, axillary, and inguinal regions.

Banti's disease and hepatic cirrhosis present similar pictures, but in the former the splenic enlargement is primary and of long duration before the changes in the liver and the resulting jaundice and ascites make their appearance. A history of alcoholism is of diagnostic importance.

vi. Splenic Tumor with Polycythæmia and Cyanosis.

Osler's Disease; Erythræmia.

Osler and others have recently described a condition characterized by cyanosis, an increase in the number of the red blood-corpuscles to 7,000,000 or even 13,000,000 to the cmm., and enlargement of the spleen. Headache, giddiness, and constipation are common symptoms. The cause of the disease is wholly unknown. It occurs in adults. The cyanosis is more marked in cold than in warm weather. Urinary changes, as low specific gravity, a trace of albumin, and hyaline and granular casts, are common. Arterial hypertension is not a constant symptom. The blood is thick and darker in color than normal. The erythrocytes vary in number at different periods in the same case. Normoblasts may be present; rarely megaloblasts. The hæmoglobin is much increased; the color-index usually about 1. The leucocytes are increased. The coagulation time is sometimes shortened. The condition is a persistent one. Such causes of cyanosis as emphysema, congenital and acquired heart disease, pulmonary sclerosis, and acetanilid poisoning must be excluded in making the diagnosis. The polycythæmia observed in those who have resided at high altitudes must also be borne in mind.

vii. Splenic Capsulitis—Perisplenic Peritonitis.

Inflammation of the capsule of the spleen occurs in arteriosclerotic atrophy of the organ, and in particular in the senile form, in acute splenitis, infarction, and abscess. It is met with also in local and general peritonitis and in chronic proliferative peritonitis. In many cases the capsular perisplenitis results from the extension of inflammation from neighboring organs, as the fundus of the stomach, the pancreas, the loops of intestine, and adhesions between these structures and the spleen are found.

Symptoms.—The clinical manifestations are often subordinate to those of the associated disease, as in gastric ulcer, pancreatic disease, or general peritonitis. In other cases circumscribed pain, tenderness, and swelling are associated with fever and much general disturbance of health.

The prognosis depends upon the nature of the primary disease. Capsular thickening and adhesions are frequently found upon post-mortem examination in cases in which no history of acute symptoms has been obtained.

viii. Infarct of the Spleen.

Embolism of the terminal branches of the splenic artery may occur in endocarditis, thrombosis of the left heart, and atheroma of the thoracic aorta. The infarcts thus caused may be single or multiple. They vary greatly in size. They are pyramidal in shape, the apex presenting toward the helium and the base toward the periphery of the organ, the correspond-

ing capsule being very frequently the seat of a circumscribed plastic inflammation. The infarct, hemorrhagic at first, may undergo softening or a gradual cicatricial change which results in a contracting and pigmented scar. Infected emboli undergo softening with abscess formation.

Symptoms.—A chill, sudden, severe pain and tenderness in the region of the spleen, which at the same time becomes enlarged, sometimes vomiting and collapse symptoms, constitute the symptom-complex. When these symptoms occur in endocarditis or in cases in which there are signs pointing to atheroma, the diagnosis of splenic infarct becomes highly probable. If there are coincident indications of renal infarct, such as severe pain referred to the loins, and hemorrhagic and albuminous urine, the diagnosis is positive. A friction sound may be heard. In many cases in which the anatomical diagnosis is made neither symptoms nor the signs of enlargement of the spleen were present during life.

ix. Suppurative Splenitis—Abscess of the Spleen.

This condition results from direct infection by pyogenic micro-organisms. There are two forms: simple and embolic. The former may result from traumatism or be secondary to infective processes in adjacent structures or infection by way of the blood. The latter occurs in septic conditions, the infected emboli being derived from local suppurative foci in distant parts. The simple abscess is usually single. It may vary in size from a cherry to the dimensions of the enlarged and overdistended spleen, the substance of which it replaces. Embolic abscesses are small and numerous, and begin as infarcts which rapidly undergo softening and are converted into collections of pus.

Symptoms.—The symptoms are those of infarction, namely, localized pain and tenderness, splenic enlargement, and irregular fever. They develop rapidly in embolic, more gradually in simple, abscess. There may be cough and dyspnœa due to interference with the movement of the diaphragm; gastric derangement caused by sepsis; œdema of the lower extremities arising from compression of the abdominal veins.

Physical Signs.—The lower end of the spleen may be palpated below the margin of the ribs. Fluctuation may be elicited. The rupture of the abscess may be followed by sudden pain and collapse symptoms, and the discharge of a considerable quantity of pus by vomiting or by the bowel, or in rare instances by way of the bronchi or the urinary passages.

Prognosis.—A great majority of the cases die. Recovery may take place by the resorption of the fluid contents of a small cavity and inspissation, or after the evacuation of the pus spontaneously or by a surgical operation.

x. Rupture of the Spleen.

Spontaneous rupture must be extremely rare. It is said to occur in connection with the acute enlargement of the organ in enteric and malarial fevers. Undue force in palpation or some similar traumatism may be suspected. Rupture of the spleen has been noted in childbirth. Severe blows, contusions, or penetrating wounds have caused rupture of the normal

spleen. The rupture may occur at the site of an infarct, or an abscess may give way. The symptoms denote sudden internal hemorrhage associated with intense pain in the splenic region. There may be extended dulness in the splenic region or in the flanks due to collections of blood. The abdomen may be swollen and the seat of general distress.

The **diagnosis** becomes probable when, in the presence of acute or chronic enlargement of the spleen, or in the case of direct violence to the left side of the trunk, sudden intense local pain, collapse symptoms, and pallor arise, together with signs of increasing enlargement of the spleen or accumulation of fluid in the flanks. An exploratory laparotomy should be performed without delay.

Prognosis.—In a few instances spontaneous recovery has resulted, as shown by the cicatrix when death has occurred at a remote period after the accident. Most of the cases are promptly fatal. Immediate operation has been the means of saving life. Localized tumors of the spleen, the so-called splenic adenomata, which consists of localized hyperplasias of splenic tissue within the spleen itself, fibromata, gummata, primary and secondary sarcomata, hydatid and other cysts are rare conditions mostly of pathological rather than clinical interest.

XIII.

THE DIAGNOSIS OF DISEASES OF THE GLANDS OF INTERNAL SECRETION.

I. GENERAL CONSIDERATIONS.

Recent physiological studies have shown that in addition to the nervous control of the various functions of the body there are chemical substances produced in certain organs, the glands of internal secretion or the endocrine glands, which, passing into the blood, are carried to distant organs upon the functions of which they exert an influence of great importance. To these substances has been given the name of chemical messengers or harmones.

The normal action of these chemical substances is to regulate the functions of the organs or tissues which they influence and thus contribute to and maintain the welfare of the body. Derangements of function of the endocrine glands may take the form of (1) hypofunction, in consequence of destruction of anatomical elements or surgical ablation of the whole or part of the gland, with relief of the symptoms following the administration of glandular substance or transplantation of portions of the gland derived from an animal source; or (2) hyperfunction, resulting from increased functional activity as the result of hyperplastic or other changes, and relieved by the operative removal of a portion of the gland in question; or (3) dysfunction, in which abnormal changes in the quality of the internal secretion may have occurred or a disproportional activity between parts of its substance having different physiological properties as in the case of the hypophysis cerebri.

These derangements of function after a time lead to corresponding derangements of function in other endocrine glands—multiglandular syndromes—a fact which warrants the hypothesis of a "harmonopoietic system."

There are glands which have no external secretion or duct, as the thymus, thyroid, parathyroids, the hypophysis, the epiphysis, and the adrenals, and glands that elaborate not only an internal secretion that enters the blood, but also an external secretion that finds its way directly into other organs or receptacles by means of an excretory duct, as the liver, pancreas, and genital glands.

II. DISEASES OF THE THYROID GLAND.

i. Acute Thyroiditis.

Acute inflammation of the thyroid gland is rare. It may follow traumatism. Much more commonly it occurs in association with an acute infectious disease, as enteric fever, scarlet fever, diphtheria, rheumatic fever, pneumonia, or mumps. In a case recently under my observation it developed in the course of an attack of influenza. In very rare instances acute thyroiditis has been noted as a primary affection.

Symptoms.—The whole gland may be affected, or only one lobe. The attack begins with a chill or chilliness followed by high fever. Swelling, pain, and tenderness rapidly develop. Externally there may be redness with engorgement of the superficial veins, and cyanosis. Internal pressure upon the blood-vessels, œsophagus, and trachea causes headache, dysphagia, dyspnœa, and stridor. Œdema of the glottis may occur. As a rule, resolution takes place in the course of several days or a week or two. Occasionally suppuration occurs. Destruction of the entire gland has been followed by myxœdema which has sometimes been observed after an attack of acute uncomplicated thyroiditis.

ii. Goitre—Bronchocele.

Definition.—Hypertrophy of the thyroid gland. This term includes all enlargements of the thyroid gland not caused by inflammation, new growths, tuberculosis, syphilis, Graves's disease, and animal parasites. The anatomical varieties of goitre are, (a) parenchymatous, (b) vascular, and (c) cystic.

Goitre occurs as a sporadic and as an endemic disease.

1. Sporadic Goitre.—The temporary congestions which give rise to enlargement of the thyroid body in girls at puberty, in many women during menstruation, and in some during pregnancy cannot be regarded as goitres. Nor do the transient swellings caused by the pressure of a tight collar or excessive use of the voice constitute goitre. Enlargement of the gland due to parenchymatous, vascular, or cystic lesions, and more or less persistent, is not uncommon and occurs almost without exception in the female sex. Age is a predisposing factor of some importance. There are rare cases of congenital goitre. The disease is uncommon in

childhood. It frequently first appears after puberty or in early adult life, but may not appear until fifty or later.

2. Endemic Goitre.—This affection is prevalent in circumscribed regions in many parts of the world. These regions are frequently but not exclusively mountainous, or deep valleys surrounded by mountains. Sometimes they are plains, especially in lake countries. Parts of Switzerland, the southern slopes of the Italian Alps, the Himalayas, the hill country of China, and some regions in Siberia are seats of endemic goitre, either alone or in association with cretinism. The disease is rare in North America. It occurs in some parts of Pennsylvania, the parts about the eastern end of Lake Ontario, and in the Province of Quebec.

The exciting cause of endemic goitre is supposed to be contained in the drinking water of goitrous districts. This opinion is supported by the following facts, which are generally accepted:

i. A healthy family coming to reside in a goitrous district presently may develop goitre among its members.

ii. Drinking water from a new and distant source has been followed by subsidence of goitre.

iii. An outbreak has followed the introduction of water from a goitrous region.

iv. Certain wells in Europe have had the reputation of causing goitre in those habitually drinking their waters.

Symptoms.—The enlargement may involve the entire gland, or only one lobe. Moderate-sized goitres cause no annoyance beyond that due to the deformity which they produce. Large tumors may give rise to dyspnœa by pressure upon the trachea; small tumors extending beneath the sternum may compress the veins. In extreme cases the goitre may compress the œsophagus and give rise to difficulty in deglutition. Sudden death has occurred in large goitres from pressure upon the vagi, or hemorrhage into the substance of the gland or the adjacent tissues.

Accessory Thyroids; Parathyroids.—These may lie in the thyroid or near it. Their number varies, rarely, however, exceeding four. In some instances thyroid tissue is situated at the root of the tongue, in the mediastinum, or even in the pleural cavity. A lingual thyroid may exist in the substance of the tongue or attached to the hyoid bone.

Diagnosis.—Direct.—The disease may be readily recognized. It is an important characteristic of all tumors of the thyroid gland that they move upward in deglutition.

Differential.—Goitre is to be discriminated from, (a) adenomata, simple or malignant; (b) malignant neoplasmata, both carcinomatous and sarcomatous. The important diagnostic criteria are the smooth parenchymatous enlargement involving one lobe or the entire gland; the uniform vascular enlargement with distinct varix arrangement, pulsation, and murmur; or recognizable agglomerate cyst formation.

Prognosis.—Treatment is not usually satisfactory. Iodine and the iodides, ergot, and counterirritants, much recommended, are not always successful. Thyroid extract is of questionable value. Large goitres may be removed.

iii. Exophthalmic Goitre.

Hyperthyrea; Graves's Disease; Basedow's or Parry's Disease.

Definition.—A disease caused by derangement of the internal secretion of the thyroid body, and characterized by exophthalmus, enlargement of the thyroid, tachycardia, and tremor.

Pathology.—Various views in regard to the essential nature of this affection have been from time to time entertained, but that which now has the most satisfactory basis of support is that it is a primary disease of the thyroid gland, resulting in an increased or deranged internal secretion causing hyperexcitability of the vegetative nervous systems, functional derangements of other glands of internal secretion, and disturbances of metabolism. In defense of this view the following facts are adduced:

i. The active proliferation in glandular substance during the progress of the disease.

ii. The production of symptoms resembling those of exophthalmic goitre by the administration of thyroid extract.

iii. The fact that thyroid extract usually aggravates the symptoms of the disease.

Etiology.—Predisposing Influences.—*Sex.*—The disease is common in women; comparatively rare in men. *Age.*—The onset usually occurs in early adult life, somewhere between the eighteenth and fortieth years. Rare cases have been observed in infancy. *Heredity.*—It sometimes occurs in several members of the same family, and has been observed in three successive generations. Infections as influenza and acute rheumatic fever appear to act as predisposing influences in local infections in the upper air passages. In rarer instances the symptoms have followed the prolonged administration of iodine.

Exciting Cause.—Acute infections, especially tonsillitis, rheumatic fever, and influenza, have been noted in many instances shortly before the onset of exophthalmic goitre. More significant are severe depressing emotions, worry, anxiety, fright, and overfatigue as antecedent conditions and possible causal factors. There is a close resemblance between the immediate effects of sudden fright or terror and the symptoms of exophthalmic goitre, namely, exophthalmus, tachycardia, and tremor.

Symptoms.—The cardinal symptoms have been mentioned in the definition, namely, exophthalmus, goitre, overaction of the heart, and tremor. With these are usually associated anæmia, emaciation, sweating, diarrhœa, and irregular or suppressed menstruation. The principal clinical phenomena vary in degree and in the order of their development.

1. Exophthalmus.—In some instances the protrusion is so great as to prevent closure of the eyelids. In others it is so slight as to be scarcely noticeable. It is frequently different in degree upon the two sides and sometimes distinctly unilateral. Commonly a rim of white is seen above and below the cornea. In rare instances the eyes may be dislocated from their sockets.

Graefe's Sign.—When the eyeball is moved downward the upper lid does not follow it as in normal conditions. *Dalrymple's Sign.*—The palpe-

bral fissure is wider than normal, owing to spasmodic retraction of the upper lid. *Stellwag's Sign.*—Infrequent, irregular, and incomplete winking. *Moebius's Sign.*—Insufficient power of convergence for near objects. The foregoing signs are commonly associated. *Joffroy's Signs.*—When the head is bowed forward and the patient asked to look up without changing his posture, the forehead is not wrinkled as occurs in health.

FIG. 334.—Exophthalmic goitre.—German Hospital

Pupillary changes and retinal lesions are rare. Defects of vision are uncommon. Subjective ocular symptoms, as sensations of pressure and phosphenes, occur. Ulceration of the cornea may take place, and in extreme cases destruction of the eyeball. Pulsation of the retinal arteries is frequent.

2. ENLARGEMENT OF THE THYROID BODY.—The enlargement is usually moderate. Usually both lobes are affected, as a rule unsymmetrically, and more commonly the right to a greater extent than the left. The swollen gland is generally soft, but may be dense and hard, especially when goitre has preceded the disease. Distinct expansile pulsation, thrill, and a systolic or continuous murmur are common phenomena. Sometimes the murmur is double. The thyroid enlargement undergoes remarkable fluctuations in volume, increasing for a time and then subsiding, or undergoing repeated changes in the course of a short time. The attention of the patient is often first called to this symptom by the tightness of the neckband or collar. Slight enlargement of the adjacent lymph-glands frequently occurs.

FIG. 335.—Exophthalmic goitre.—German Hospital.

3. CIRCULATORY DERANGEMENTS.—This group of symptoms constitutes the most constant and striking features of the disease. Tachycardia is always present. The pulse-frequency varies from 90 to 100 beats in the minute in the early course of the disease, to 100 to 130 and even 140 to 160 in severe and advanced cases. The rate is usually increased upon effort and under the influence of emotion. Forcible pulsations of the vessels at the root of the neck are associated with increased cardiac action and a greatly extended visible impulse. As a rule the patients complain of palpitation and sometimes feel the sensation widely over the body. In some cases the over-action of the heart causes little discomfort. The heart is almost always dilated and sometimes hypertrophied. Its action

is, as a rule, regular, but in grave cases arrhythmia is often marked. Sinus arrhythmia, premature contractions, auricular flutter, auricular fibrillation, heart-block, occur as manifestations of myocardial degeneration. Systolic murmurs are common at the apex and across the base of the heart. The heart sounds are often intense, and may, in some instances, be heard at some distance from the body of the patient. Acute dilatation of the heart may occur. There may be visible pulsation of the peripheral arteries and the pulse-beat may be felt in the palms and finger-tips. A capillary pulse is common and the venous pulsation in the back of the hand may often be seen. Flushing of the neck and face is often pronounced. Other vasomotor derangements are pruritus, urticaria and dermographism.

4. Tremor.—This cardinal symptom is a variable one. It may be forcible and annoying, involving not only the extremities but also the head, or so slight as to be discovered only upon careful examination. It is entirely involuntary, of limited extent, and about eight or nine to the second. It is usually symmetrical, but may be more marked upon one side, or confined to, or more distinct in, a single limb. A coarser tremor sometimes occurs during excitement.

Anæmia, emaciation, and loss of strength are common. Fever is rare, but subjective sensations of heat, and copious perspirations, are common symptoms. Vomiting and diarrhœa occur in the absence of obvious cause. The electrical resistance is diminished, a fact attributed to the moisture of the skin due to vasomotor dilatation. Pigmentation is not uncommon. The parts chiefly affected are the face, neck, trunk, nipples, and flexures of the arms and thighs. In rare instances there is a general bronzing like that of Addison's disease: in other cases an irregular patchy discoloration. Patches of leucoderma may appear. Transient œdema is common and myxœdema is occasionally seen. The nutrition of the hair suffers and complete alopecia may occur. The teeth sometimes undergo rapid decay. Albuminuria, glycosuria, and true diabetes are occasionally encountered in the course of the disease. Menstrual derangements are common. When pregnancy occurs the condition of the patient often improves and the fœtus is born at term. Individuals suffering from Graves's disease are frequently the subjects of incompletely developed genital organs. This occurs in both sexes and has been attributed to an associated involvement of other endocrine glands—thymus, gonads.

Nervous and mental symptoms are very common. Insomnia is a common symptom. Tremors, cramps of the hands and feet, a sensation of giving way at the knees, and increased tendon reflexes are observed. Irritability, altered disposition, and mental depression occur in most of the cases. Various psychoses, phobias and obsessions, manic-depressive states, dementia-præcox, and paranoia are encountered as complications. The acute mania which sometimes precedes death has been attributed to sudden, intense thyroid intoxication.

Course and Duration.—Incomplete forms are common and often overlooked. They may be characterized by tachycardia, nervous irritability, slight tremor, and little enlargement of the thyroid or protrusion of the eyes. There are acute and chronic forms. The latter are more common. Acute

cases usually occur in childhood. They may last a few days or several weeks and get well. Relapses may occur. As a rule, when in adults the disease is well marked, recovery is infrequent. Death occurs from intercurrent affections.

Diagnosis.—When the four cardinal symptoms are associated a positive diagnosis can be made. Difficulty may arise in the rudimentary forms. Absence of exophthalmus or of thyroid enlargement is not often complete. These symptoms when slight are, in association with tremor and tachycardia, most significant. Anæmia, pigmentation, emaciation, and mental changes aid the diagnosis. Atypical cases are often overlooked.

Prognosis.—The disease is essentially chronic. Acute cases are exceptional. A guarded prognosis should be given. In individual cases the more urgent the symptoms the less favorable the outlook. When the symptoms are mild the prospect of recovery is proportionately better. Cases in which the onset is sudden and severe, after fright, sometimes recover in a short time.

Krumbhaar, as a result of electrocardiographic studies in 51 cases of goitre, mostly of the toxic type, 47 of which submitted to surgical operation, arrives at the following conclusions:

1. In early cases of toxic goitre the characteristic tachycardia is not accompanied by any signs of myocardial change that are demonstrable with the string galvanometer. 2. With persisting overaction of the heart, hypertrophy of either ventricle may become manifest. 3. Progressive hypertrophy and overaction result in myocardial degeneration that may be manifested by any type of cardiac irregularity: sinus arrhythmia, premature contractions, auricular flutter, auricular fibrillation, heart-block, etc. 4. If the existing intoxication is the chief factor in the production of the arrhythmia, this may disappear with removal of the intoxication. 5. Successful treatment, whether medical or surgical, improves the cardiac condition by this means. This is shown not only by the occasional disappearance of an arrhythmia but also by diminution in the size of the *T* wave and in the pulse-pressure as well as by the general clinical condition. 6. The development of diphasic or inverted *T* waves, especially in Leads I and II, should probably be considered as influencing prognosis unfavorably. (*Am. Jr. Med. Sciences,* Feb., 1918.)

iv. Myxœdema.

Hypothyroidism; Athyroidism.

Definition.—A constitutional disease caused by the impairment or loss of the function of the thyroid gland, and characterized anatomically by absence, atrophy, or goitrous degeneration of the thyroid, and clinically by profound nutritional changes, a firm, inelastic swelling of the skin and subcutaneous tissues, and nervous and mental symptoms.

Varieties.—Three forms are recognized: the infantile, or cretinism; the adult, or myxœdema proper; and postoperative myxœdema, or cachexia strumipriva.

Etiology.—The sporadic form of cretinism is due to the absence of the thyroid or suppression of its function. The endemic form occurs under local conditions associated with goitre, and is encountered in parts of France, Switzerland, and Northern Italy. The myxœdema of adults may develop at any period of life from puberty to seventy. More than half the cases begin between thirty and fifty. The disease is more common in females than in males in the ratio of about 6 to 1. In certain families myxœdema has been observed in two generations and there are numerous reports of its occurrence in several members of the same family. With reference to its geographical distribution, it is comparatively common in cold climates and very rare in the tropics. More cases have been observed in Great Britain and Europe than elsewhere. Many cases have been recognized in America. It is uncommon in Philadelphia. The colored races are said not to suffer from the affection. No walk of life is exempt, though it appears to be more common among the poor, a fact probably explained by the relatively larger numbers constituting this class. It is more frequent among married women and especially among those who have borne children than in others and has been thought to have some relation to the menopause, since many of the cases begin about the age at which the child-bearing function ceases. Occasionally it has followed symptoms of exophthalmic goitre. Postoperative myxœdema follows the total extirpation of the thyroid. The cases of thyroidectomy to which cachexia has not supervened are thought to have been incomplete, an unobserved portion of the gland having remained.

Fig. 336.—Cretinism; female, 9 years old.—Rotch.

The symptoms of cretinism differ from those of the adult form, but those of the latter and of the postoperative form are identical.

1. Cretinism.—This affection occurs as a sporadic and as an endemic disease. There is retardation of physical and mental development. The condition is not usually recognized until toward the end of the first year, but becomes completely developed in the course of the second year. At this time the clinical picture is characteristic. The face is large, round, and bloated; the eyelids are puffy and congested; the nose is flattened and the alæ are thickened and coarse; the lips are full and swollen; the tongue is large and protrudes from the mouth. There is constant drooling. The eruption of the teeth is delayed, and they soon become carious. The complexion is pasty and sallow; the expression dull and fatuous. The fontanelles

remain open. The belly is protuberant, the hands and feet are clumsy and ill-formed, the legs short, the muscles weak and flabby, and the child is unable to stand or walk. The hair is thin and brittle and the skin dry and harsh. The rectal temperature is commonly subnormal and there is great sensitiveness to cold. The mental condition remains undeveloped. Speech is acquired late and is rudimentary. There are various degrees of idiocy. Older cretins are dull and amiable, not often vicious, and present many of the characteristics of infancy at the age of ten or fifteen years. The fatty tumors seen in the myxœdema of adults are very common.

Diagnosis.—The DIRECT DIAGNOSIS is unattended with difficulty. The facies, the retardation of physical and mental development, the fat pads, and the subnormal temperature are distinctive.

DIFFERENTIAL DIAGNOSIS.—The early cases are sometimes mistaken for rickets. In the latter affection sweating of the head, craniotabes,

a *b*

FIG. 337.—*a*, adult type of myxœdema. *b*, six weeks later; a reduction in weight of 20 pounds under thyroid therapy.—Jefferson Hospital.

restlessness at night in the early stages, and special deformities, as the rosary, enlargement of the epiphyses at the wrist and ankles, and bow-legs in the later stages, are characteristic. *Juvenile Cretinism.*—The forms that begin at the age of five or later in children previously well nourished and healthy, in consequence of atrophic changes in the thyroid following an acute febrile infection, are comparatively rare. Occasional cases of transient mild cretinism are seen in children in the second or third year and may be ascribed to functional derangements of the thyroid.

2. Myxœdema of Adults; Gull's Disease.—As a rule, the disease develops insidiously; exceptionally in the case of young adults it may be recognizable in the course of a few weeks. Languor, subjective sensations of cold, tardiness of movement, change of expression due to myxœdematous infiltration of the subcutaneous tissues of the face, and increase in the size and weight of the body are early symptoms.

The following phenomena are characteristic of the fully developed affection: (a) Dense, inelastic swelling of the skin and subcutaneous tissues,

which does not pit upon pressure. This swelling is general but is most marked in parts where the subcutaneous tissues are loose. It is frequently first noticed in the face, sometimes in the lower extremities and the backs of the hands. (b) A change in the facies due to the swelling, which obliterates the lines of expression. The eyelids are swollen; the upper eyelid tends to droop; the eyebrows are habitually elevated; the forehead is corrugated by deep transverse wrinkles; the nose thickened and enlarged; the cheeks are full, large, sometimes pendulous, and often the seat of a circumscribed, pinkish flush. The lips are thickened and coarse and the mouth appears to be enlarged. Similar changes are seen in the ears and the parts about the angles of the jaw. (c) The hands and fingers are swollen and lose their expressiveness, assuming a thick, flat shape which has been described as "spade-like." Similar changes take place in the feet. (d) A general increase in the size and weight of the body, which may be mistaken for obesity, but which differs from that condition in the distribution of the swelling and the texture of the tissues. (e) Local swellings of the skin and subcutaneous tissues, especially in the supraclavicular regions and in the posterior aspect of the neck. The occasional occurrence of fibrofatty pads in the retroclavicular spaces in healthy persons is to be borne in mind. (f) Changes in the thyroid gland, which cannot be felt at all or is of uncertain size in many of the cases, distinctly atrophic in others, and normal or increased in size in a very small proportion. (g) Dryness and roughness of the skin, thinness and brittleness of the hair, and alopecia which affects not only the scalp but the brows and axillary and pudendal regions. Similar atrophic changes affect the nails, which become cracked and discolored, while the teeth undergo rapid caries and become loose. (h) Subnormal temperature. The range is often continuously a degree or more of Fahrenheit's scale below normal and frequently several degrees. Temperatures of 95°–93° F. (35°–34° C.) or even lower have often been observed. Remarkable falls to 77° F. and 66° F. (25°–19° C.) have occurred before death. (i) Muscular weakness and slowness of voluntary movements. (j) Mental changes, especially slowness of apprehension and response, impairment of memory, sensitiveness, and irritability. The speech is tardy and drawling. Hallucinations are common. Fixed delusions may develop, and insanity terminating in dementia occurs. Albuminuria and glycosuria occasionally are noted.

3. **Postoperative Myxœdema; Cachexia Strumipriva.**—The symptoms are those of the common form in adults.

Diagnosis.—The diagnosis is unattended with difficulty. From renal or cardiorenal dropsy myxœdema is to be differentiated by the character of the œdema, its failure to pit upon pressure, the absence of renal and cardiac lesions, the small thyroid, low temperature, the mental symptoms, and the promptly remedial effect of thyroid extract. From ordinary obesity the diagnosis is readily made.

Prognosis.—Formerly the outlook was practically hopeless. The patients improved somewhat in warm weather, but became worse as the cooler season approached. The course of the disease was chronic and progressive, sometimes extending over a period of ten or fifteen years. Death was due to intercurrent disease, very often to tuberculosis. At present in a ma-

jority of instances the prognosis is favorable. Treatment by thyroid extract causes the symptoms to disappear in a few months, and the continued administration of this remedial principle maintains the improvement.

III. DISEASES OF THE PARATHYROID GLANDS.

We have no knowledge of the effects of an increased activity of the parathyroid glands. It was early observed that complete extirpation of the thyroid gland was followed in some subjects by symptoms of myxœdema; in others by acute convulsive phenomena. These varying results were found to depend upon the amount of parathyroid tissue removed with the thyroid and that the ablation of all the parathyroid tissue with the least possible amount of thyroid resulted in a characteristic convulsive condition corresponding to tetany and the tetanoid states.

Hypoparathyreosis; Status Parathyreoprivus.—These terms have been suggested by Halsted to designate degrees of the cachexia caused by the removal of some or all of the parathyroid bodies. It may also be caused by the arrest of the blood supply of those glandules in ligation of the thyroid arteries in partial thyroidectomy. Cachexia thyreopriva in many of the cases has been a complex condition made up of thyroid and parathyroid privation.

Tetany.—Among the symptoms of cachexia parathyreopriva tetany occupies the first place. This varies in degree from a subtetanic condition to the most violent manifestations of postoperative tetany, often terminating in death. Bleeding, with infusion of salt solution into the veins, the subcutaneous or intravenous injection of an extract or emulsion of the parathyroid glands, and finally the injection of a nucleoproteid prepared by Beebe from an emulsion of parathyroids, are followed by temporary relief of the tetany in parathyroidectomized animals, and favorable results in human beings have been reported by several observers abroad and by Halsted in one case in this country. Better and lasting results may be confidently expected from the transplantation or implantation of the living parathyroid gland. These glandules appear to play an important part in calcium metabolism, since their removal is followed by an increased excretion of calcium and diminution of the calcium content of the tissues. In dogs suffering from the most violent postoperative tetany, with muscular rigidity, clonic spasm, extremely rapid respiration and pulse, all the symptoms can be instantly dispelled by the injection of a calcium salt, the acetate or lactate, in 0.5 gramme doses, into the jugular vein (MacCallum).

Tetany is characterized by tonic, cramp-like spasms, especially in the fingers, hands, and upper limbs. It occurs in epidemics in some countries, especially in and about Vienna, Northern Italy and Sweden. The cause is obscure. Tetany may be of infectious origin, and it is also seen in ergotism, in the diarrhœa and rickets of young children, in nursing women, after extirpation of the thyroid gland, and in dilatation of the stomach. It may occur at any time of the year, but the epidemic prevalence is most frequent during the months of January, February and March. Chvostek advanced the theory that the real cause is a defective action of the parathyroid glands.

The tetany and tetanoid states occurring in non-operative cases are doubtless due to temporary or more or less prolonged parathyroid insufficiency, since the identity of these forms of the disease and the postoperative states has been established. According to the statistics of Fränkl-Hochwart, shoemakers and tailors seem peculiarly vulnerable. The disease is rare in America.

Symptoms.—The tetanic spasms are oftenest seen in the small muscles of the fingers and hands, causing flexion of the basal phalanges, extension of the distal phalanges, and turning in of the thumb. This is sometimes called the "obstetric hand." The wrist and elbow are flexed. The toes may be similarly affected; also the calf muscles.

The spasms are often painful, and usually intermittent. They can sometimes be excited by pressure on the nerve-trunks or main vessels—Trousseau's sign.

There is increase of the mechanical and electrical irritability. Tapping on nerve-trunks causes a lively contraction (Chvostek's sign), often well marked in the face; and the galvanic current, even of mild power, causes active, even tetanic, responses, which may increase, rather than diminish, with repeated stimulation (Erb's sign). The nerves may be hypersensitive to pressure.

Other muscles, such as those of the trunk, tongue, and respiration, are involved in severe cases, and the ocular muscles, according to Kunn, may be affected. Nystagmus has been seen. Other ocular symptoms noted by a few observers, and which are evidently very rare, if they belong to the disease at all, are neuroretinitis, optic atrophy, unequal pupils, diplopia, cramp of the eye-muscles, especially blepharospasm, reddening of the conjunctivæ, and lachrymation. Slight paresis and anæsthesia (or hypæsthesia) are present in the limbs after severe attacks of the cramps.

There is no affection of consciousness, as a rule, but in severe cases constitutional reaction, such as fever, accelerated pulse, etc., is seen.

The carpopedal spasms and laryngospasms of infancy are manifestations of tetany. In many cases infantile convulsions (eclampsia) are due to the same cause. The reflexes show no constant changes.

Sometimes the disease ends fatally, especially when caused by some of the more serious conditions mentioned above. A patient in the Philadelphia Hospital died of exhaustion and pulmonary œdema; but the prognosis appears to be good in the majority of simple cases.

Diagnosis.—The tetanic symptoms are so characteristic that the diagnosis is easy.

The cramps seen in chronic uræmia are not to be mistaken for tetany.

The French describe an hysterical type of tetany, but it cannot be made to cover all cases; when the disease is truly hysterical the mental state and the presence of other stigmata suggest the right diagnosis, and such signs as Trousseau's and Chvostek's are wanting.

It is probable that a variety of affections cause tetanoid symptoms, and further study is necessary to shed light on these obscure cases. Tetany has no relation to true tetanus, and does not resemble it.

IV. DISEASES OF THE PITUITARY BODY.

Dyspituitarism.

The symptoms may be due to an overactivity of the functions of the gland or an under activity. Clinically the symptoms usually attributed to increased internal secretion are frequently associated with symptoms due to diminished secretion. The hypothesis that some forms of disease of the pituitary body are due to a perversion rather than an increase or decrease of its secretion has support.

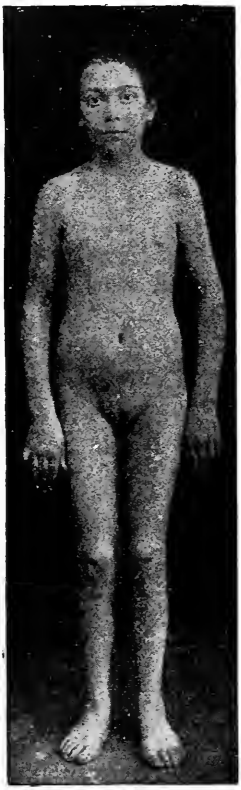


FIG. 338.—A typical case of infantilism with an enlarged 2 cm. sella. Patient aged 20 years 6 months: height 4 ft. 4 in. (133 cm.) (Ettore Levi).*

Our knowledge of this subject is largely due to the labors of Cushing and his associates.[1] This author as the result of experimental and clinical studies has arranged the cases into five groups as follows:

GROUP I.—Cases of dyspituitarism in which not only the signs indicating distortion of neighboring structures but also the symptoms betraying the effects of altered glandular activity are outspoken.

GROUP II.—Cases in which the neighborhood manifestations are pronounced but the glandular symptoms are absent or inconspicuous.

GROUP III.—Cases in which neighborhood manifestations are absent or inconspicuous though glandular symptoms are pronounced and unmistakable.

GROUP IV.—Cases in which obvious distant cerebral lesions are accompanied by symptomatic indications of secondary pituitary involvement.

GROUP V.—Cases with a multiglandular syndrome in which the functional disturbances on the part of the hypophysis are merely one, and not a predominant feature of a general involvement of the ductless glands.

Neighborhood symptoms comprise such local pressure disturbances as primary optic atrophy, constriction of the visual fields, oculomotor palsies, anosmia, sometimes signs of implication of the crura cerebri and the uncinate region, epistaxis and pharyngeal protrusions.

Glandular manifestations consist of modifications of skeletal development, as overgrowth—acromegaly, gigantism—undergrowth—infantilism, dwarfism; cutaneous and subcutaneous changes, as hypertrophy of papillæ, hypertrichosis, thickening of subcutaneous tissues with slight bogginess or œdema in hyperpituitarism, and in hypopituitarism smoothness, transparency, freedom from moisture, with small, thin nails and scanty pubic and axillary hair and more or less irregular pigmentation, adiposity, high sugar tolerance, subnormal temperature, slow pulse, drowsiness, asthenia and arterial hypertension.

*Figures 338, 339, 340, 341, 342 are inserted by courtesy of Dr. Harvey Cushing.

[1] The Pituitary Body and Its Disorders. J. B. Lippincott Company, 1912.

Psychic disturbances may be neighborhood symptoms due to pressure distortion of the temporal or frontal lobes by a growth, and consist of

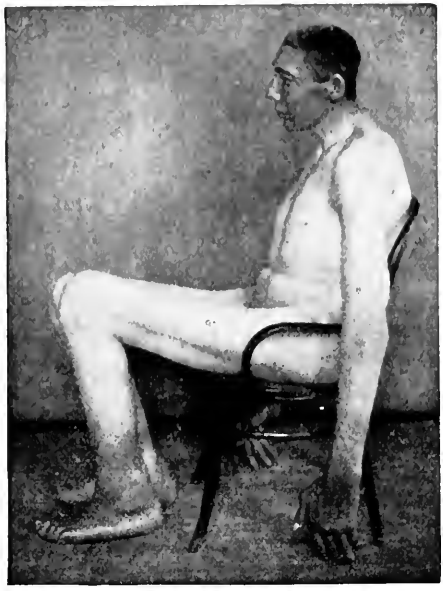


FIG. 339.—Preadolescent hyperpituitarism with giant overgrowth. Note extreme length of arms and thighs in relation to usual 18-inch chair.

FIG. 340. —Characteristic square hand. Note heaping up of tissues about nails. "Type en large" of Marie.

changes in disposition, loss of memory, disorientation, untidiness, and lack of appreciation of the existing condition; or they may be primarily glandular symptoms, as wakefulness, lack of decision, irritability and distrust. In

hyperpituitarism or in hypopituitarism any grade of mental disturbance from a mild psychosis to grave mental derangements with epilepsy may occur.

Retarded sexual development is on the one hand very common when the manifestations of hypophyseal lesions antedate puberty, while upon the other hand amenorrhœa or impotence with retrogressive sexual changes usually occurs when dyspituitarism develops after adolescence.

The excessive subcutaneous fat observed in a large proportion of the cases has led to the employment of such descriptive terms as adiposis dolorosa, adiposis universalis, dystrophy, dystrophia adiposo-genitalis, adiposis cerebralis, to designate the affection.

Examination by X-rays may show distortion or enlargement of the sella.

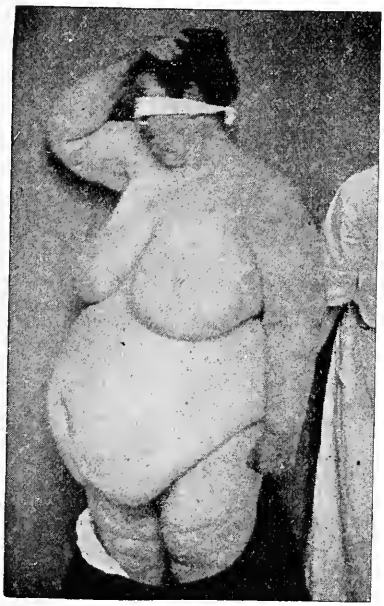


Fig. 341.—Extreme obesity—302 pounds ("adiposis dolorosa"), attributable to hypopituitarism.

ACROMEGALY.

This clinical manifestation is so common and well characterized that its separate consideration is important. The hypothesis that the skeletal changes are due to a hyperplasia of the pars anterior of the hypophysis with the production of an altered or excessive secretion has much support in the collected facts.

Definition— A trophic disease characterized by symmetrical overgrowth in the soft parts and bones of the face and extremities, with deformities of the spinal column and thorax, cerebral and sexual symptoms and evidences of the implication of other glands of internal secretion.

Etiology.—Predisposing Influences.—It is a disease of early adult life, most of the cases having first shown themselves in the third decade. No case has been noted at an earlier age than twenty, and in only a few has the onset occurred after thirty-five. Males and females are equally liable, with the exception that in cases beginning at a late period more women suffer. The disease is not uncommon, and its geographical distribution is wide. Many of those who suffer from acromegaly were persons of previously large growth.

The Exciting Cause.—This is not known. The coarse lesions found after death consist of tumors of epithelial origin, cysts, hemorrhages and sclerotic conditions. There are cases in which neither tumors nor histological changes have been present.

Symptoms.—The disease develops gradually, the early symptoms consisting of lassitude and vague pains, and abnormal sensations—*parœsthesiæ*—in the head and extremities. Amenorrhœa and impotence are very common. The characteristic anatomical changes are first noticed in the face. The lines of expression are altered and the countenance is distorted.

The skull is enlarged; the supercilliary ridges are very marked and prominent; the zygomatic arches protrude, and there is remarkable hypertrophy of the upper and lower jaw bones, the latter projecting in a conspicuous manner. The alveolar processes are similarly enlarged and the teeth are separated. The soft parts undergo corresponding and even more marked changes. The eyebrows are bushy and thick and almost meet in

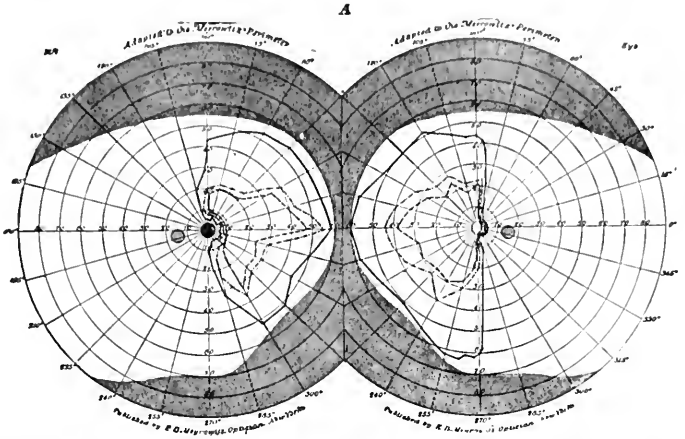


FIG. 342A.—Case X of Cushing's Series. Fields on admission. Bilateral primary optic atrophy. Bitemporal hemianopsia including the macula on left and sparing it on right. Acuity: Left 5/50; right 15/50. Marked photophobia. No exophthalmos; no diplopia.

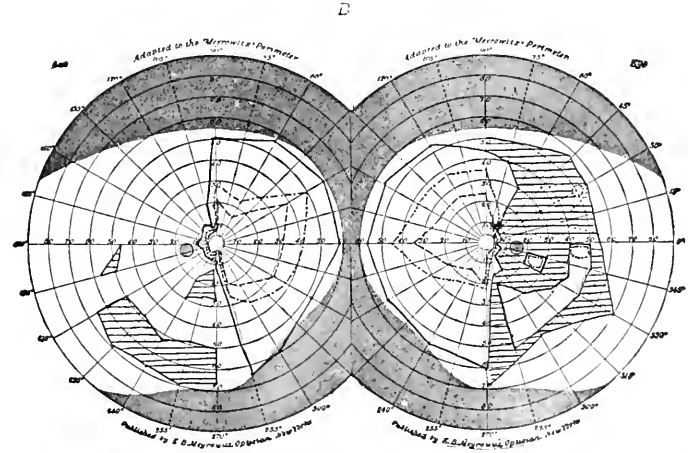


FIG. 342B.—Case X. Fields 3 months after operation. Acuity: Left 15/30; right 15/20. Photophobia has disappeared.

the median line; the eyelids are greatly thickened; the nose is conspicuously enlarged; the ears are enormously hypertrophied; and the lips swollen. Some degree of exophthalmus is often present and may vary in extent from time to time. Enlargement of the tongue is a very common lesion. This organ attains in some cases such dimensions that it is impossible to close the mouth. Similar and progressively increasing deformities involve the hands and feet, and are rendered especially noticeable by the relatively small size of the arms and legs, which do not share to any marked extent

in the enlargement of the extremities. The thickening affects alike the bones and soft parts, and while not greatly interfering with the functions of the hands causes the remarkable appearance described as spade-like. The nails are broadened but not incurvated and the drum-stick bulbous enlargement of pulmonary osteo-arthropathy is not seen. The feet are generally enlarged, the great toe being especially increased in size. The skin of the affected parts commonly preserves its natural appearance. In some instances, however, it becomes coarse and pigmented. As the disease advances the spine becomes affected. There are hyperostoses and exostoses of the vertebral processes, ankylosis of the vertebræ, and kyphosis. The clavicles are enlarged and there is a gradual enlargement of the ribs. The thyroid body may be atrophied or hypertrophied, but such changes are not constant. There are cases in which the thymus is enlarged. In a very large proportion of autopsies—73 in 77—the hypophysis cerebri has been found to be affected. Very often it has been increased in size, sometimes to that of an egg. Sometimes the increase is in an upward, sometimes in a downward direction. The histological changes are not constant. Glandular hyperplasia, softening, cystic degeneration, and fibrosis are described. Many of the cases have been regarded as malignant. Symptoms suggestive of cerebral tumor, namely, headache, vertigo, somnolence, are not infrequent. Glycosuria is common; actual diabetes by no means infrequent: diabetic coma is an occasional terminal event. Albuminuria is rare. Ocular symptoms, bitemporal hemianopia, optic atrophy, and oculomotor palsies occur. The great frequency with which lesions of the hypophysis have been found lends support to the hypothesis that this organ is the source of an internal secretion by which the growth of the body is regulated, and that acromegaly is the result of some vitiation or defect in that secretion.

Fig. 343. — Acromegaly; diabetes mellitus; in a woman aged 46.—Jefferson Hospital.

The brain symptoms are very often those of cerebral tumor and in many cases there are, in addition, the evidences of abnormalities in the functions of other organs of internal secretion, thyroid, thymus, pancreas.

Diagnosis.—The direct diagnosis of acromegaly is unattended with difficulty. In no other affection do similar anatomical changes in the bones and soft parts occur. Difficulty arises in earliest periods of the disease. The differential diagnosis from gigantism depends upon the specific nature of the changes in acromegaly, and the fact that they are for a long time chiefly confined to the face and extremities, the vertebral column and thorax being later involved. It is nevertheless true that acromegaly frequently develops in persons of large frame. From the rare cases of progressive overgrowth of one member or a part of it, or of one side of the body, acromegaly is to be distinguished by the fact that the hypertrophies are symmetrical and

chiefly confined for a long period to the face and extremities. Paget's disease, lentiasis, rickets and myxœdema are to be considered. Röntgenography should be employed in the study of every case.

Prognosis.—The disease may run a chronic, progressive course of a score or more of years. There are cases which terminate in death in four or five years. Restoration to normal conditions does not occur. A cachexia may develop. A majority of the cases die of some intercurrent affection, as diabetes, cancer, croupous pneumonia or phthiasis pulmonalis.

Dystrophia Adiposo-genitalis.

Frohlich's Syndrome.

Definition.—A disease due to a tumor or other lesion of the hypophysis cerebri, causing a decrease in the functional activity of that endocrine organ especially of the posterior lobe.

Symptoms.—The symptoms are those of brain tumor and manifest themselves as (a) general symptoms due to increased intracranial pressure, headache usually bitemporal or frontal, not rarely diffuse; vomiting and vertigo; psychic phenomena and epileptiform convulsions. Or they may be (b) neighborhood symptoms due to local pressure, visual derangements usually bilateral, primary optic atrophy, choked disk, hemianopsia, diplopia; spastic paralysis, derangements of the heat-regulating mechanism; (c) naso-pharyngeal disorders, epistaxis, prevalent splenoidal sinusitis, polyp formations, the presence of the tumor mass in the naso-pharynx.

The röntgenological examination reveals changes in the sella turcica and sometimes also in the body of the sphenoid bone; rarely foci of calcification in the tumor itself. Other characteristic symptoms are caused by diminished internal secretion of the hypophesis. These comprise excessive fat accumulations either general or locally over the abdomen and hips; changes in the growth and distribution of the hair, baldness, loss of eyebrows and axillary and pubic hair and a reversal of the upper line of the pubic hair in the sexes, that of the male being transverse and that of the female triangular with the apex extending toward the umbilicus. If the disease begins before puberty the genitalia retain the characteristics of the infantile type and remain undeveloped. In males early development of this dystrophy is followed by changes in the skeleton which reverts to the feminine type, not only in the pelvis but also in the extremities, the fingers being slender and tapering. Diabetes insipidus is common, but diabetes mellitus is rare, the carbohydrate tolerance being commonly increased. The asthenia, lowered blood-pressure, habitual high pulse frequency, abnormal pigmentations, and other changes in the skin have been attributed to associated derangements of the functions of other endocrine glands—multiglandular syndromes.

Diagnosis.—In typical cases there is little difficulty. The association of obesity, retarded development or atrophy of the genital organs and bitemporal hemianopia is characteristic, the X-ray picture confirmative.

Early symptoms and atypical forms are obscure. Contraction of the field on the temporal sides, evidences of intracranial pressure, menstrual derangements, the rapid accumulation of fat, and a high output of non-saccharine urine are suggestive. The examination by X-rays is imperative.

V. DISEASES OF THE EPIPHYSIS CEREBRI OR PINEAL GLAND.

The functions of the pineal gland are not well understood. Experimental researches have yielded conflicting results. Tumors and cysts occur but they are not very common. In many of these cases there is an internal hydrocephalus, the result of venous compression. In children, especially in boys, such growths may be accompanied by the so-called pineal syndrome, consisting of rapid increase in stature, precocious mental development, early sexual maturity and obesity. Whether this symptom-complex is due to derangement of an internal secretion of the pineal gland or to disturbances in neighboring structures which lead to abnormal action of other endocrine glands is unknown. The symptoms of the hydrocephalus are those of intracranial pressure, headache, vertigo, vomiting and drowsiness. Choked discs may be present. Optic atrophy is not uncommon. Various palsies of the eye-muscles occur. Nystagmus and exophthalmus have been observed. The occasional occurrence of polyphagia, polydipsia and polyuria has been reported. While obesity is common, emaciation with cachexia occasionally occurs. The diagnosis depends upon the presence of the pineal syndrome in association with the symptoms and signs of a tumor in the mid-brain region occurring in early life.

VI. DISEASES OF THE ADRENAL BODIES.

The adrenal glands are composed of a medullary and a cortical substance.

1. The medulla consists largely of nervous elements derived originally from sympathetic nerve cells and corresponding in development, structure and physiological properties with accumulations of similar tissue that lie in the neighborhood of the abdominal sympathetic ganglia. These structures, the cells of which stain deeply with the chrome salts, have on that account received collectively the name of the "chromaffin tissue system." All these collections of tissue yield on extraction a blood-pressure raising substance similar to epinephrin (adrenalin).

2. The cortex is composed of epithelial cells and has its developmental origin in a mass of tissue adjacent to the kidneys in which the sex glands have their origin. In the lower animals these cell-collections remain separate from the suprarenals, but in the higher vertebrates, including man, these epithelial structures form the substance of the cortex. Minute accessory collections are found in and about the pelvis of the kidney, the epididymis, the prostate and along the spermatic veins. To the position of the tissues from which the cortex develops and the histological relationship of the accessory masses the collective designation "interrenal tissue system" owes its use. The functions of this tissue system are not clearly understood.

General Considerations.—The symptomatology of disease of the suprarenal bodies is obscure. Marked lesions of these organs have been found at autopsy in cases in which, during life, no symptoms suggestive of any disease involving them have been observed. In another group of cases tumor, pressure symptoms, and lumbar and sacral pain have suggested

disease of these bodies. Again, metastatic growths in various organs have been ascribed to malignant disease in the suprarenals. Finally, a characteristic symptom-complex—Addison's disease—has been found to be associated in a large proportion of the cases, but not in all, with definite lesions of these organs.

Addison's Disease.

Definition.—A constitutional disease, due to modification or cessation of the internal secretion of the adrenal glands in consequence of destructive lesions, usually tuberculous, and characterized by asthenia, gastro-intestinal irritability, and pigmentation of the skin.

Etiology.—Predisposing Influences.—Addison's disease is a rare affection. The most common and important predisposing influence is tuberculosis. It is somewhat more common in males than in females. It may occur at any period of life, but less frequently before twenty and after sixty than in the intervening stages. Malaria, alcoholism, depressing emotions, exposure, and traumatism have been regarded as causes of this as of many other diseases. In so far as they predispose to tuberculous infection they may act in this way.

Morbid Anatomy.—Very common are tuberculous deposits with caseous changes. Comparatively rare are atrophy,—either simple or resulting from chronic interstitial changes,—malignant disease, and interstitial hemorrhage. In a small group of cases the organs have been found normal, but inflammatory or pressure changes have been present in the semilunar ganglia. Cicatricial tissue implicating the semilunar ganglia and adrenals, together with sclerotic and pigmentary changes in the nerves, is not uncommon. The thyroid gland in the absence of cancerous infiltration is usually small; the thymus sometimes persistent; the spleen enlarged or the seat of amyloid change.

Fig. 344.—Addison's disease; showing distribution of bronzing.

Pathology.—Two principal hypotheses have been advanced: 1. That Addison's disease is an affection of the abdominal sympathetic system caused by disease involving the suprarenal bodies, or the solar plexus or semilunar ganglia. 2. That it is the result of loss of the function of the suprarenals. The theory of an internal secretion essential to normal metabolism is now fully established.

Symptoms.—The onset is usually insidious. Little has been added to the description of Addison,—"Anæmia, general languor or debility, remark-

able feebleness of the heart's action, irritability of the stomach, and a peculiar change of color in the skin." This description may be somewhat amplified.

1. Asthenia.—The first symptom is commonly a sense of fatigue in the performance of every-day accustomed duties. Fatigue symptoms, at first intermittent, soon become constant. Weakness is both muscular and circulatory. This may be marked, while the muscles still feel firm and the general nutrition and weight are preserved. The cardiac asthenia may be paroxysmal and lead to attacks of vertigo or syncope. The blood-pressure is low. Headache is common and pain in the loins may be an early and suggestive symptom.

The examination of the blood has yielded variable results. Anæmia is by no means a constant phenomenon. Eosinophilia is often present, and occasionally lymphocytosis.

Mental dulness is frequently observed.

2. Disturbances of the Digestive Organs.—These appear gradually. Anorexia, epigastric distress, nausea and vomiting, and attacks of diarrhœa without obvious cause occur with varying prominence in a majority of the cases throughout the course of the disease. In a small proportion of the cases they are absent. Epigastric and abdominal pain are common toward the end. Gastro-intestinal symptoms may occur early.

3. Pigmentation of the Skin.—Sooner or later a dark pigmentation of the skin appears. In many cases this is the first symptom to attract attention. The pigment accumulation is gradual. The affected portions of the skin are at first yellowish- or grayish-brown and later become brown or even blackish. The discoloration in well-marked cases is diffuse but never uniform. It usually begins in the parts exposed to the light and in those normally the seat of pigment deposits, and is deepest in those areas and in regions subjected to the habitual pressure of the clothing. The face, backs of the hands, nipples and their areolæ, the genitalia, axillary folds, and parts pressed by the waistband, garters, and collar are especially pigmented. There may be diffuse patches of deep discoloration with indistinct borders, or small dark pigment areas with well-defined borders upon a less deeply pigmented surface. Patches of leucoderma are occasionally seen. The mucous membranes of the lips and mouth are frequently the seat of an irregular patchy pigmentation. Less commonly a similar discoloration affects the conjunctivæ or the vaginal mucosa. The course of the disease is essentially chronic, and marked by normal or subnormal temperature, subjective sensations of cold, suppression of menstruation, and the gradual development of cachexia. Urinary changes are inconstant. Increased pigments have been observed. Dropsy is rare. Death may occur early in the disease from sudden syncope. More commonly it is the result of progressive asthenia or advancing tuberculous lesions. It is sometimes preceded by acute toxæmic phenomena, urgent vomiting and diarrhœa, delirium with motor excitement, and convulsions followed by coma.

Diagnosis.—Direct.—A positive diagnosis may be made from the association of the following symptoms: general languor and debility, remarkable feebleness of the heart's action, irritability of the stomach and

irregular diarrhœa, a peculiar pigmentation of the skin and mucous membranes. In the early stages the diagnosis may be impossible. It is to be borne in mind that Addison's disease may occur without pigmentation.

The mere presence of pigmentation does not, however, warrant a diagnosis of Addison's disease. It may occur in the following conditions: physiological peculiarities due to racial and climatic influence; accidental pigmentation of the skin and mucous membranes in persons otherwise healthy; the mild cutaneous pigmentation of aged persons; chronic malaria; various cachectic states, especially those due to cancer and tuberculosis; pellagra; leukæmia; as the result of scratching in various chronic skin diseases attended by itching—prurigo, eczema, phthiriasis; diffuse melanosarcoma of the skin; rare cases of exophthalmic goitre; scleroderma; pregnancy and uterine disease; hæmochromatosis such as may occur in rare cases in association with hypertrophic cirrhosis of the liver and diabetes mellitus—*diabète bronzé*; certain cases of pancreatic disease; chronic arsenical poisoning; and argyria. Chronic jaundice has been confounded with the pigmentation of Addison's disease, an error that ought not to occur. Much difficulty attends the recognition of the actual condition when jaundice develops in the course of the disease and is associated with abnormal pigmentation.

Differential.—In a doubtful case the foregoing conditions must be successively excluded as the cause of pigmentation by the systematic application of the rules of diagnosis, and the presence of asthenia, cardiac weakness, and gastro-intestinal symptoms determined before a positive diagnosis is made. In view of the tuberculous nature of the suprarenal disease in the majority of the cases the tuberculin test may be employed.

Prognosis.—The disease is probably always fatal. The rare reported recoveries have been ascribed to errors in diagnosis, or the mistaking of a prolonged remission such as sometimes occurs in the very chronic cases for an actual recovery from the disease. Cases unattended by bronzing of the skin are often rapidly fatal. The average duration is about two years. There are exceptional cases of very long duration—ten to thirteen years.

Conditions Due to Increased Function of the Cortex of the Suprarenal Body.

Hyperplastic conditions and tumors (nephromata) of the suprarenal cortex occur. They are sometimes associated with hyperfunction of the interrenal tissue system which results in changes in the endocrine parts of the sexual glands with extraordinary developmental perversions of the secondary sex-characters. These anomalies are much more common in females than in males. There are prenatal, acquired juvenile and adult forms. In the prenatal form the external genitalia resemble the male type, while the internal sexual organs are those of the female—pseudohermaphroditism; in the juvenile form, the external genitalia appear to be normal at birth and throughout infancy but about the fifth or sixth year, or indeed earlier, the changes in the sex-organs and the general body are those which normally occur at puberty. Enlargements of the mammæ occur in girls and menstrua-

tion is established and in boys sexual excitement, erections and pollutions. The adult form occurs mostly in females after menstruation has been fully established for some time and is characterized by menstrual irregularity, obesity, increased muscular development and physical strength with mental excitability and marked sexual propensities. These changes are accompanied by hypertrophy of the external genitalia and an abnormal growth and distribution of hair upon the body and face. Later, mental and physical depression, emaciation, albuminuria, and low arterial pressure occur and are the precursors of death. The diagnosis of interrenal disease is probable from the above conditions and is rendered positive by the recognition of a tumor in the region of a kidney (nephroma) and metastatic growths.

VII. DISEASES OF THE THYMUS GLAND.

The functions of this transitional organ are unknown. It has been accredited with a hypothetic internal secretion capable of antagonizing infections, a hypothesis which accounts for the comparative immunity of young infants against many of the acute febrile infections. The weight of the thymus gland at birth is variously estimated at from 8 to 13 grammes. It gradually increases in size till the end of the second year, from which period it undergoes progressive atrophy, until at puberty it is a shrivelled mass containing only traces of its original structure.

I. **Persistence of the Thymus.**—This occurs under varied conditions which may be casual or accidental, but are not understood. It is usually a post-mortem finding, but may be suspected when localized dulness along the sternal border on the left side from the second to the fourth rib may be made out. Persistent thymus has been frequently observed in exophthalmic goitre.

II. **Hypertrophy.**—Enlargement of the thymus has been regarded as the cause of thymic asthma or laryngismus stridulus—a condition attributed to the pressure of the enlarged organ. Dyspnœa, laryngeal cough, and bronchial râles in young infants have been caused by hypertrophy of the thymus and relieved by partial excision, but that it is the cause of laryngismus stridulus is by no means established. Sudden death—*mors thymica*—may occur in lymphatism in infants with hypertrophy of the thymus. The children are found dead in bed or die in a short time with symptoms of asphyxia. In certain cases of sudden death in adults the thymus has been found greatly enlarged with signs of status lymphaticus. Hypertrophy of the thymus has been occasionally present in epilepsy.

III. **Atrophy.**—Primary atrophy of the thymus in infants may be attended with general atrophy in the absence of other symptoms. Secondary atrophy attends tuberculous and other wasting diseases.

IV. **Hemorrhages.**—Extravasations of blood in the substance of the gland have been frequently found in children dead of asphyxia.

V. **Tumors of** the thymus are sarcoma, lymphosarcoma, dermoid and other cysts, and gumma. Miliary tuberculosis and gumma are rare pathological findings. Mediastinal tumors frequently have their origin in the thymus.

The majority of the diseases of the thymus gland cannot be recognized during life, and are of pathological rather than clinical interest.

VIII. STATUS THYMICO LYMPHATICUS; LYMPHATISM; CONSTITUTIO LYMPHATICA.

Definition.—A constitutional condition characterized by hyperplasia of the lymph-nodes and lymphatic tissues generally, the spleen, the thymus, and the bone-marrow, and the liability to sudden death.

Morbid Anatomy.—The lymph-nodes of the pharynx, thorax, and abdomen are chiefly affected. The superficial glands of the cervical, axillary, and inguinal regions may be involved. As a rule, the enlargement is moderate in degree and symmetrical, and differs in this respect from that of Hodgkin's disease. The enlargement of the spleen is moderate, the thymus is persistent and usually hypertrophied, and the yellow marrow of the long bones may be replaced by red marrow. The thyroid body is enlarged, and hypoplasia of the heart and aorta is present. Many of the subjects are rhachitic.

Etiology.—The condition has been variously ascribed to heredity and to over-irritability of the lymphatic tissue. It occurs principally in children and young persons. The association with rickets is probably accidental.

Clinical Phenomena.—The patients are fat and flabby, and have pallid, opaque skins. Hypertrophy of the tonsils and overgrowth of the pharyngeal adenoid tissue, swelling of the thyroid, overgrowth of the thymus (as indicated by dulness over the manubrium sterni, especially at its left border), enlargement of the superficial lymph-nodes, and especially signs of enlargement of the mesenteric glands, are present. Moderate enlargement of the spleen is suggestive.

Diagnosis.—When the foregoing signs are well marked the diagnosis may be made with some confidence, but a positive diagnosis cannot often be affirmed. The number of cases in which the lesions of lymphatism have been found after unlooked-for death, under circumstances in which such an event has appeared unaccountable—as the injection of diphtheria antitoxin serum for prophylactic purposes, chloroform or ether anæsthesia for trifling operations in young children, during the bathing of young children wholly without apparent cause, and occasionally during convalescence from the acute febrile infections—have given the condition a sinister significance. This fatal occurrence has been attributed to the pressure of the hypertrophied thymus upon the trachea, or to a perverted or excessive internal thymus secretion—lymphotoxæmia.

The DIFFERENTIAL DIAGNOSIS from Hodgkin's disease rests upon the more marked and asymmetrical enlargement of the superficial glands, and the absence of hypertrophy of the pharyngeal lymph structures in the latter affection; from glandular fever, upon the acute and self-limited course of the latter disease, the presence of fever, and the subsidence of the enlargement of the lymph-nodes after defervescence.

Prognosis.—The subjects of lymphatism have feeble powers of resistance, and in consequence of the hypoplasia of the heart and aorta are especially liable to sudden death.

IX. DISEASES DUE TO DERANGEMENTS OF THE ENDOCRINE FUNCTIONS OF THE SEX GLANDS.

The physiological functions of the sex-glands are threefold. They not only produce ova and spermatozoa which correspond to the external secretions of other glands having tubular ducts, but they also elaborate chemical substances which are discharged into the blood-stream and exert a powerful influence upon development and temperament. The groups which produce the internal secretions of the ovaries are the luetin cells; those in the testicles are the interstitial cells of Leydig. Hyperfunction may be caused by hypertrophy or malignant disease of the organs. Precocious puberty results at a very early age with the physical signs and psychic manifestations peculiar to the sexes respectively. This form of hypergenitalism occurs as a secondary manifestation in diseases of other endocrine glands as the epiphysis cerebri and the interrenal system.

Hypofunction of the sex-glands is associated with degenerative changes in these organs. Castration in early life is followed in the male by almost complete failure of development in the sexual characteristics. Such individuals are true eunuchs. The eunuchoids are persons who have not been castrated or spayed, but nevertheless present undeveloped sex-organs and bodily configuration resembling that of the true eunuch. Not only the gonads, but also other endocrine glands are involved and the condition constitutes one of the forms of multiglandular syndrome. There are two groups of cases, those in whom the condition is manifest in early life and those in whom the sex-organs have fully developed with normal functions, but have also undergone atrophic changes with corresponding somatic and psychic signs of hypogenitalism. In such subjects of either sex sterility and absence of sexual desire are almost constant symptomatic phenomena.

The menopause, whether natural or artificial, is very often associated with nervousness, pains in the extremities, subjective sensations of heat and cold, attacks of faintness, anxiety and apprehension, digestive derangements, constipation and rapid increase of fat.

X. GIGANTISM; INFANTILISM.

Gigantism.—The skeletal changes in acromegaly, particularly those affecting the long bones, are the essential feature of gigantism. Most of those suffering from acromegaly are taller than normal. Hyperpituitarism in childhood causes gigantism, but hyperpituitarism developing in adult life does not cause gigantism. It is probable that changes in the functions of the pituitary body constitute an etiological factor in overgrowth, since a very large proportion of giants develop acromegaly.

Infantilism.—A condition in which there is an incomplete arrest of development at an early stage with retention of the bodily and mental characteristics of infancy in early childhood. The developmental fault involves the genital organs and secondary sexual characters, so that puberty does not occur. Incomplete involution of the lymphatic system, delayed growth and ossification, and retention of the relative proportions of the body and extremities peculiar to infancy are essential features of the con-

dition. Infantilism occurs in both sexes, but is much more common in females. For the male it is sometimes described under the term *feminism*. Occasionally it is associated with early tuberculosis, but more frequently with disease of the endocrine glands, particularly the thyroid and pituitary. In the latter infantilism involving the genitals and sexual characters occurs in persons of great stature (infantilism with gigantism). The same association is seen in true eunuchs and certain eunuchoids.

XI. THE MULTIGLANDULAR SYNDROMES.

Interrelationships of the Endocrine Glands.

Derangement of function in respect of increased or diminished activity in the thyroid and parathyroids, adrenals, thymus, pancreatic islets, pituitary, testis and ovary is associated with reciprocal functional and anatomical changes in other and frequently all of the correlated organs. To the resulting condition the term "multiglandular syndrome" is applied. It is not always clear which of the glandular organs is primarily at fault. There are cases in which at autopsy sclerotic and atrophic processes involving to the same extent several of the glands of the internal secretion have been found. The glands most frequently involved are the thyroid, the hypophysis, the adrenals and the testicles and ovaries. Cirrhosis of the liver and chronic pancreatitis have also in some instances been present.

Among the more common multiglandular syndromes described are acromegaly with exophthalmic goitre, exophthalmic goitre with Addison's disease, and myxœdema and Addison's disease with sex-organ derangements. These facts lend support to the view that disease of an endocrine gland is only in rare instances limited to that gland, but that it commonly represents a dominant element in a multiglandular complex. It is also probable that in the interaction among these organs alike in health and disease inhibitory as well as accelerating influences may be exerted.

XIV.

THE DIAGNOSIS OF DISEASES OF THE CIRCULATORY SYSTEM.

DISEASES OF THE HEART.

I. ABNORMAL POSITIONS OF THE HEART.

Congenital displacement is rare. It occurs in general transversus viscerum. The apex lies to the right; the right ventricle toward the anterior chest wall; the left ventricle behind it; while the pulmonary artery lies at the right border and the aorta at the left border of the sternum. The condition is readily distinguished from displacements by the electrocardiogram, which is lead for lead reversed. Transposition of the heart alone—*dextrocardia*—is usually associated with complicated developmental anomalies affecting both the arteries and chambers. The heart is displaced

to the left in congenital arrest of development of the left lung. Children with complete defect of the anterior chest wall and abdomen with protrusion of the heart—*ectopia cordis*—are usually stillborn or die shortly after birth. When there is merely a fissure of the sternum caused by defective development of the manubrium and body, the heart remains in its normal position and may be studied through the overlying soft tissues.

Acquired displacements are far more common and of greater diagnostic significance. Sudden death which sometimes occurs in massive pleural effusions has been ascribed to abrupt, angular bending or twisting of the inferior vena cava. The upward displacement of the heart, which occurs in the retraction of the lungs in patients long bed-ridden, or in meteorism, ascites, pregnancy, and large abdominal tumors, may lead to an erroneous diagnosis of dilatation of the heart. The high position of the apex and of the inferior border of the lung are significant. Large subphrenic abscesses displace the heart toward the opposite side and thus increase the resemblance to empyema, while a massive spleen, as in leukæmia, displaces the apex upward and outward.

The displacements of the heart toward the sound side in pleural effusions and pneumothorax, and toward the affected side in the contraction of the lung after resorption of a large pleural exudate, or in fibroid phthisis, are too familiar to lead to diagnostic errors. The fact that the displaced heart, in consequence of adhesions, occasionally remains upon the sound side is less generally understood. In pulmonary emphysema the dislocation of the heart is downward. Aneurisms of the ascending and transverse portions of the arch of the aorta displace the heart downward and toward the left.

II. GENERAL CONSIDERATIONS.

Much of our present knowledge of diseases of the heart is the outcome of recent physiological research and the use of instruments of precision and graphic methods. In fact, the sphygmograph, the polygraph, and the electrocardiograph have not only revolutionized the older conceptions of the anatomy and physiology of the heart but they have also brought about a new era in the consideration of cardiac diseases from the standpoint of the clinic. That these mechanical devices have wholly superseded the older diagnostic methods can, however, no more be admitted than that the newer views have rendered the older facts of descriptive anatomy and pathology obsolete. The sphygmograph as at first used for taking and recording independent radial pulse-tracings, notwithstanding the enormous time and energy expended in attempts to establish its employment in clinical medicine, is no longer used except in polygraphic instruments which record simultaneously the cardiac movements, the arterial pulse and the venous pulse. Finally, the electrocardiograph in bringing to light many new facts in regard to the origin and conduction of cardiac motor impulses has simplified the subject of the cardiac arrhythmias and greatly facilitated their diagnosis. Many of the facts revealed by this instrument were first brought to light by the polygraph and may still be obtained by its use, but at a much greater expenditure of time and effort. A distinguished clinician has observed that "If we

except the usefulness of venous pulse-tracings for the diagnosis of tricuspid insufficiency and of pulsus alternans, electrocardiography has relegated sphygomography to a place of scarcely more than historical interest." But these instruments, in the words of Mackenzie, "are not available to the general practitioner," except at the heart station of a well-equipped clinical laboratory.[1]

III. THE FUNCTIONAL AFFECTIONS OF THE HEART.

Definition.—The functional affections of the heart include those sensory and motor derangements which occur in the absence of demonstrable anatomical changes in the organ.

The qualifying adjective "functional" is used in its common sense, to denote the absence of anatomical lesions demonstrable during life or after death. It is appropriately employed in this connection to designate, not lesions of the heart itself, but rather disorders of its innervation. Hence these affections are also properly spoken of as cardiac neuroses.

It is important to observe that all the morbid phenomena noted in functional disorders may attend the structural diseases of the heart.

The functional affections of the heart are:

A. Sensory:
   Subjective sensations referred to the precordia.
   (a) Heart consciousness.
   (b) Precordial distress.
   (c) Precordial pain.

B. Motor: Derangements of rhythm.
   (a) Arrhythmia.
   (b) Rapid heart—tachycardia.
   (c) Slow heart—bradycardia (brachycardia).
   (d) Momentary arrest—syncope.

C. Motor and sensory combined:
   Palpitation.

Etiology.—PREDISPOSING INFLUENCES.—These comprise, (a) a weak and delicate organization associated with an impressionable nervous system; (b) anæmic conditions; (c) lithæmia and allied derangements of metabolism and excretion; and (d) morbid conditions directly affecting the nervous system, as organic diseases of the brain and cord, chorea, epilepsy, and the acute and chronic infections.

Certain of these conditions are inherited, others acquired. Thus the descendants of nervous or insane parents, those begotten of elderly persons, those who have in infancy been exposed to privation and neglect, or who have suffered from serious or protracted disease, are especially prone to functional disturbances of the heart. To a less extent is this true of gouty, tuberculous, and syphilitic persons.

Functional derangements of the heart are much less frequent in childhood than in adult life.

The EXCITING CAUSES include: (A) those acting upon the nervous system, (a) directly, as intense mental emotion, fear, anger, grief; (b) reflexly,

[1] See articles on The Polygraph and The Electrocardiograph, Vol. 1.

as gastro-intestinal irritation, intestinal parasites, or foreign bodies in the intestinal canal; or acute or chronic general or focal infections; and (B) those acting, by means of mechanical disturbance of the circulation, upon the heart, as violent exercise or exertion.

In a large proportion of the cases disorder of function occurs in the absence of demonstrable cause.

Symptoms.—In general terms the symptoms of the functional disorders of the heart consist in abnormal sensations referred to the precordia and derangement of the motor functions. These sensory and motor derangements are not always associated. More commonly the movement of the heart is deranged, its action being accelerated, retarded, or irregular, without abnormal distress or pain or a sense of oppression; and in comparatively rare instances precordial pain occurs in the absence of motor disturbance.

When the functional disorder is paroxysmal or of a high grade of intensity, it is usually accompanied by increased frequency of respiration, and very often by pallor of the face and slight cyanosis. Especially is pallor associated with syncope.

When the derangement is not paroxysmal, but persistent, the rhythm of the respiration is not usually disturbed.

Physical examination yields a limited number of definite signs. The frequency of the heart's action and the degree and character of the arrhythmia may be recognized upon palpation. By this method of examination we also detect, especially on palpation, the change in the character of the impulse. We observe also by this means and by inspection whether or not the impulse is extended. We determine by the position of the apex beat, and may confirm the observation by percussion, that the heart is not enlarged. Upon auscultation the first sound is found to be sharp and valvular and shortened in duration, while the second sound remains distinct or is accentuated. In very rapidly acting or very irregular hearts transient murmurs, usually mitral systolic, are common.

A. Sensory.—Subjective sensations referred to the precordia.

(a) Heart Consciousness.—Neurotic and neurasthenic individuals frequently complain of disagreeable sensations in the region of the heart. They feel the extra-systole and the compensatory pause, and use such phrases as "the heart stumbled," or "the heart turned over," to express the sensation. But in many cases of arrhythmia there are no subjective sensations. It is a mistake to call the attention of patients to arrhythmia or intermissions of which they are unaware, since the consciousness of such irregularities is often a cause of great distress. Sometimes these sensations take the form of weight or oppression on lying on the left side and are relieved on turning upon the back or right side.

(b) Distress.—Precordial distress is common in neurotic individuals, both in functional and organic disease of the heart. It is extreme in the overacting heart of violent exertion and is the essential element in palpitation. It is common in intense emotion. It may be reflex in character and is frequently due to gastro-intestinal derangements. It is often transient. Cutaneous hyperalgesia and tenderness elicited by pressure or gentle squeezing of a fold of skin in the precordial region are met with in abnormal con-

ditions of the heart varying from slight functional derangement to grave disease.

Distress is very prominent in the affection known as "irritable heart," common among soldiers and first described by Da Costa during the Civil War. Increased pulse frequency, loss of tonicity, dyspnœa and exhaustion upon effort are associated symptoms. The condition can usually be traced to some infection. In the great majority of the cases there are no organic lesions.

(e) Precordial Pain.—This neurosis is common in nervous individuals. It occurs in valvular disease of the heart, especially in aortic and mitral stenosis and the later periods of aortic insufficiency, and in the forms of myocarditis associated with sclerosis of the coronary arteries. It is the chief element in angina pectoris and an important symptom in aneurism of the aorta.

It is usually of moderate severity and is often described as a dull aching. It is, however, sometimes very severe and presents the features of a violent attack of angina pectoris. The precordial pain which is induced by effort is more commonly associated with actual disease of the heart, while that which comes on at rest is usually due to heart exhaustion from constitutional causes. But there are many exceptions to this general rule.

Angina Pectoris.

Stenocardia.

Definition.—True angina pectoris is a symptom-complex occurring in individuals who suffer from sclerotic changes involving the ascending aorta and coronary arteries, and characterized by recurrent paroxysms of agonizing pain in the precordial region, extending to the neck and arms, especially upon the left side, and often accompanied by a sense of impending death.

Etiology.—Predisposing Influences.—All conditions which tend to the production of arteriosclerosis may be looked upon as influences predisposing to angina pectoris. Age is of great importance. The great majority of the cases first declare themselves after the fiftieth year. Attacks occasionally occur at an earlier age, but they are comparatively infrequent, while the few cases which have been recorded in childhood do not militate against the general rule. Sex exerts a remarkable influence, not in itself, but in the bearing which it has on the mode of life of the individual. Heredity plays an important part. It is by no means rare for angina pectoris to occur in successive generations. To gout, syphilis, and alcohol are justly attributed casual influences of weight. Diabetics are prone to the attacks. It has occurred after influenza.

Exciting Cause.—Any exertion, especially any sudden exertion which calls upon the heart for increased effort, may bring on the attack. The effort is usually a familiar one, as hurry to catch a car, ascending a flight of stairs, or stooping over to lace the shoes. Emotional excitement is a common exciting cause. A fit of anger may precipitate a fatal attack. The paroxysm frequently follows a hearty meal. I often say to patients:

Do not hurry, do not worry, and do not eat too much. Exposure to cold is a common cause of the attack. Several of these causes are often associated, as hurry and exposure to cold, or some annoyance after a full meal. The attack seldom comes on when the patient is at rest both physically and mentally. On the other hand there are cases in which the attack occurs immediately upon waking from sleep.

The Paroxysm.—The attack begins suddenly, very often without warning, and in a few moments attains its full intensity. There are three essential phenomena: (1) Pain, commonly described as agonizing, referred to the precordium and mostly to the region beneath the manubrium. This pain radiates to the left side of the chest, to the neck, and to the ulnar distribution in the left arm. In rare cases the pain is first felt in the wrist or arm. The right side is in some instances also affected. (2) The stenocardia of Heberden. The sensation is that of being crushed in a vise, or as if the heart were being "grasped by a mailed hand." It is to this sensation that the fixation of the muscles of the patient is probably due, for at the height of the paroxysm he is usually motionless, almost rigid, while at the beginning and end he is agitated, restless, and anxious. (3) The sense of impending death. The face denotes the anguish which the patient feels. It is pallid, gray, and bathed in sweat. The pulse is often full and slow; its tension greatly increased. It may, however, be nearly normal. The heart sounds are commonly feeble, but clear, and not rarely a soft apex murmur may be heard. In the majority of patients the attack is accompanied by a marked rise in systemic blood-pressure. Cases in which such a rise does not occur are relatively serious in character. Vomiting is not unusual, and death may occur during the act. The duration of the attack does not usually exceed four or five minutes, but recurrences are common, so that the agony may last, with remissions all too brief, for several hours. This is, however, unusual. Nocturnal attacks are usually very severe and longer in duration than those which occur by day. Instant death during the attack is not uncommon. In other cases the patient may fall into a syncope and not revive. The paroxysm subsides as suddenly as it came on, often with belching, the passing of a large quantity of clear urine, exhaustion, and asthmatic symptoms. There are other cases in which the patient, while much distressed and exhausted, is, in the course of an hour or two, able to resume his occupation. The first paroxysm has in many cases proved fatal; in other cases the attacks occur at varying intervals for many years. The first attack may not be followed by a second for a period of months or years, and there are instances of a series of violent attacks followed by no recurrence for a long period.

Angina sine Dolore.—Gairdner used this term to designate "an element of subjective abnormal sensation present in cardiac diseases, which when it is not localized through the coincidence of pain is a specially indefinable and indescribable sensation." There are undoubtedly cases of heart disease in which paroxysms of profound disturbance of the nervous system referable to the heart occur in the absence of pain, but with an agonizing fear of impending death.

There are many explanations concerning the attack of angina pectoris,

among which that of paroxysmal ischæmia or intermittent claudication is the best as a working hypothesis, but none of which as yet fully meets the requirements of the conditions. Mackenzie suggests that the heart muscle induces pain on the principle of the summation of stimuli.

Diagnosis.—The direct diagnosis rests upon the association of paroxysms having the foregoing characteristics with the evidences of general arteriosclerosis and chronic myocarditis. Increased arterial tension, a history of gout, alcoholism, or syphilis, and advanced life are of the nature of corroborative evidence.

FUNCTIONAL ANGINA PECTORIS; PSEUDO-ANGINA PECTORIS.

Two principal groups are described: the neurotic and the toxic.

Neurotic Angina Pectoris.—This form is common in hysterical and neurasthenic women. It may occur at any age. The attacks are not precipitated by muscular effort or cold, but by the multitudinous worries to which such persons subject themselves, and not rarely by injudicious eating. They recur with a periodicity which is remarkable and are more common in the night, a point in which they especially differ from angina associated with coronary or aortic sclerosis. They are usually attended by nervous symptoms—flatulent distention of the stomach and belching—and are often prolonged for several hours, with continuous agitation and restlessness. There are forms in which the paroxysm is characterized by coldness and numbness of the extremities, with agonizing substernal pains, great restlessness, and a tendency to syncope—vasomotor variety.

Toxic Angina.—This form is attributed to excesses in tea, coffee, and tobacco. It includes "tobacco heart." As a rule pain is less marked than sensations of anxiety and precordial oppression, with derangements of the cardiac rhythm. Attacks occur which are characterized by vertigo, pallor, a small, tense pulse, faintness, precordial distress, perspirations, and coldness of the hands and feet. There are three groups of cases: first, the irritable heart of smokers; second, cases characterized by precordial pain, which may be persistent and severe but is not agonizing; and third, cases in which the symptoms are those of an organic angina and there are evidences of arteriosclerosis. In the first two of these groups recovery follows abstinence from the narcotic, but in the third recovery is only partial.

The **diagnosis** of functional angina pectoris depends upon the presence of the neurotic constitution, the absence of the signs of arteriosclerosis or chronic myocarditis, the character of the paroxysms, which, however severe they may be, have neither the short duration nor the essential features of angina vera. The younger age, the sex, and the different nature of the causes by which the paroxysms are excited have diagnostic significance. There are varying grades of intensity, both in organic and in functional angina pectoris, as shown in the recurring paroxysms in the same patient, and it is not the intensity but the character of the attack which is diagnostic.

There are difficulties in the classification of the cases. Some writers refer all cases of paroxysmal precordial pain from the mildest to the most severe to the single category of angina pectoris; others describe the milder attacks as *angenoid,* indicating resemblance rather than identity.

B. Motor: 1. Derangements of Rhythm.—The cardiac mechanism is now regarded as myogenic rather than neurogenic; that is inherent in the heart muscle and not due to the action of the vagi and sympathetic nerves under the influence of higher centres. Whether its contractions originate in the muscular fibres, or wholly or in part in nerve-cells imbedded in the muscles has no special bearing upon the fact that the heart muscle with its contained nerve-cells is an anatomically contracting organ.

(a) Arrhythmia.—Under normal conditions the pulse is practically regular and rhythmic, the individual pulse-waves being of like volume and following one another at equal intervals of time. Physiological derangements of rhythm are slight and transient and occur under conditions which are attended by changes in the pulse frequency. Marked disturbances of rhythm—*arrhythmia*—are nearly always pathological and have their source either in functional derangements or demonstrable lesions of the heart.

The general causes of arrhythmia are psychic or emotional, organic cerebral diseases, reflex, toxic, or changes in the heart itself.

The various forms of arrhythmia have been considered in the section on the Pulse. (See Vol. I, pp. 468 *et seq.*) It remains to discuss them from the standpoint of cardiac diagnosis.

The rhythm of the heart is controlled by impulses that arise in the sino-auricular node, which has received the name of pacemaker. This small mass of specialized tissue lies at the junction of the superior vena cava with the right auricle. The conduction system between the auricles and ventricles comprises a node of similar tissue, the auriculo-ventricular node of Tawara, with extensions into the auricles and ventricles. The extension into the ventricles is the only functional connection between the auricles and ventricles and is known as the bundle of His. This bundle divides into two branches, one passing to the right ventricle, the other piercing the intraventricular septum to reach the left ventricle. The ramifications of these main divisions are distributed to the papillary muscles and the inner surfaces of the ventricles, these terminations constituting the fibres of Purkinje.

Motor impulses arising in the sino-auricular node are transmitted by this system to all parts of the heart. But any portion of the heart-muscle possesses the inherent power of originating contractions. The normal rhythm is, however, controlled by the pacemaker by impulses which are more rapid than the rhythm of any other part of the heart. The automatic rhythm of the outlying regions is therefore not manifest under normal conditions. When in consequence of impairment of conduction the whole heart does not receive the impulse from the sino-auricular node, independent impulses may arise from the undischarged portions. When the automatic impulses from the pacemaker are slowed or the impulses in other regions are accelerated a similar transference of the function of the pacemaker occurs.

(b) Rapid Heart—The Sinus Tachycardias.—The rate of impulse formation is subject to the inhibitory control of the vagus nerves. Section of the vagi or the administration of atropine diminishes the inhibition and increases the rapidity of the sinus rhythm. Influences that are of clinical importance are exercise, due to diminution of vagus tone and continuing

throughout and after the exercise, especially marked in feeble and anæmic persons and those suffering from circulatory insufficiency; low blood-pressure; acute and chronic myocardial disease; fever, in which the high frequency depends in part upon the rise of body temperature and in part upon the action of the fever-producing toxins; and exophthalmic goitre. In the last the tachycardia is caused by some toxic principle in the internal secretion of the thyroid gland, which acts upon the myocardium and not only causes rapid action but also in severe cases produces insufficiency, dilatation and cardiac arrhythmias.

(c) Slow Heart—The Sinus Bradycardias.—Vagus stimulation is followed by slowing of the rate of impulse formation in the sino-auricular node. The slowing of the heart which occurs in acute asphyxia and rapid increase of intracranial pressure is due to stimulation of the respiratory centre in the medulla. That which characterizes the early periods of meningitis may be ascribed to the same cause. Nasal, ocular and intestinal reflexes may give rise to vagus bradycardia. The bile pigments in jaundice act in the same way. Acute increase of blood-pressure causes vagus stimulation partly as the result of direct action upon the medullary centres; partly in consequence of reflex irritation from the heart and great vessels, and is attended by slowing of the heart. The marked slowing of the heart frequently observed during the convalescence from acute febrile infections and sometimes attended by a respiratory arrhythmia is probably due to vagus irritation.

(d) Momentary Arrest; Syncope.—Faintness is common in nervous and impressionable persons. It may result from sudden shock or intense excitement. Blood loss (even when slight), profuse diarrhœa, extreme fatigue, and severe pain may cause faintness or actual syncope. Emotional children may faint at the sight of blood. A boy of seven fainted at the sight of the denuded spot upon his arm caused in vaccination. A healthy girl of eight, of shy and timid disposition, fainted at the dinner table upon being suddenly addressed by a person whom she did not know. Fainting of this kind is due to vagal stimulation.

Syncope as a result of cerebral anæmia occurs in heart block—Stokes-Adams syndrome—in which arrest of ventricular systole is attended with loss of consciousness and convulsions and in auricular flutter in those cases in which the ventricular systole suddenly corresponds to the auricular rate and the output of blood is greatly reduced. In syncope the pulse is of very low tension.

C. **Motor and Sensory Combined.**—Palpitation.—The most extreme motor disturbance of the cardiac rhythm may occur in the absence of consciousness of any cardiac irregularity, and distressing precordial sensations may, on the other hand, be experienced while the heart's action is regular and orderly. In palpitation there is irregular or forcible action, perceptible and usually distressing to the individual. This combination of motor and sensory derangements constitutes palpitation. It is of frequent occurrence in neurotic individuals, and is common at puberty, the grand climacteric, and during menstruation. It is frequently encountered in hysteria and in neurasthenia, and constitutes an important element in the symptom-

complex known as cardiac neurasthenia. It may occur in normal individuals in consequence of violent emotion. It is a symptom of irritable heart whether due to overexercise, excess in venery, or overindulgence in tea, coffee, tobacco, or alcohol; and finally, it is a common factor of cardiac insufficiency from any cause. As a rule, palpitation is more violent and distressing in irritable heart than in valvular or myocardial disease. The symptoms vary from a mere fluttering of the heart to a violent pulsation with great distress and anxiety. In the latter case the heart's action may be slow or rapid; regular or irregular. The sounds may be clear and ringing, the second accentuated, and murmurs wholly absent. In some cases there are basic or apex murmurs, which disappear when the attack is over. The attacks are of variable duration, but do not often exceed an hour or two.

Special Forms of Arrhythmia.—Sinus Arrhythmia.—In infants occasionally, but much more commonly in children between the ages of five and ten, and about puberty, the heart is found to vary in rate with the respiratory movements, being somewhat more frequent during inspiration than during expiration. This irregularity may be recognized by auscultation with the individual in the recumbent posture and becomes more marked upon slow and deep breathing. It has for this reason received the designation of respiratory irregularity; and since graphic methods show it to arise in or near the sino-auricular node and to be due to a transient slowing of the whole heart it is known as sinus arrhythmia. The cause of this form of derangement of rhythm is stimulation of the vagus by the act of respiration. It occurs in nervous adults, especially during the convalescence from the acute infectious diseases and has been observed in connection with post-febrile bradycardia. It tends to disappear in early adult life and has been called by Mackenzie the "youthful type of irregularity." The acceleration of the action of the heart by exercise, fever, or the injection of drugs which paralyze the vagus, as atropine, causes this type of arrhythmia to temporarily disappear. Mackenzie regards it as physiological and not as evidence of impairment of the heart. Its greatest importance from the clinical viewpoint arises from the fact that it is sometimes mistaken for a sign of heart disease. There are no subjective sensations attendant upon this form of irregularity of the heart.

Dropped Beats.—This is a form of sinus arrhythmia characterized by a complete failure of the whole heart to contract during the time of one or more revolutions. It has been ascribed to actual tissue changes in the sinus region, but its occasional occurrence at irregular or long intervals makes the view that it arises from vagus inhibition more probable.

Abnormal Impulses; Ectopic Beats.—Extrasystoles; Premature Contractions.—The normal rhythmic impulses arising in the sino-auricular node pass consecutively in regular order through the muscles of the auricles and ventricles. If for any reason an automatic impulse arises at a point in advance of the regular rhythmic stimulus from the pacemaker the special form of arrhythmia which occurs is known as an extrasystole or premature beat. These new and independent impulses may arise in any region of the heart muscle and are known according to their point of origin as auricular, ventricular or nodal extrasystoles.

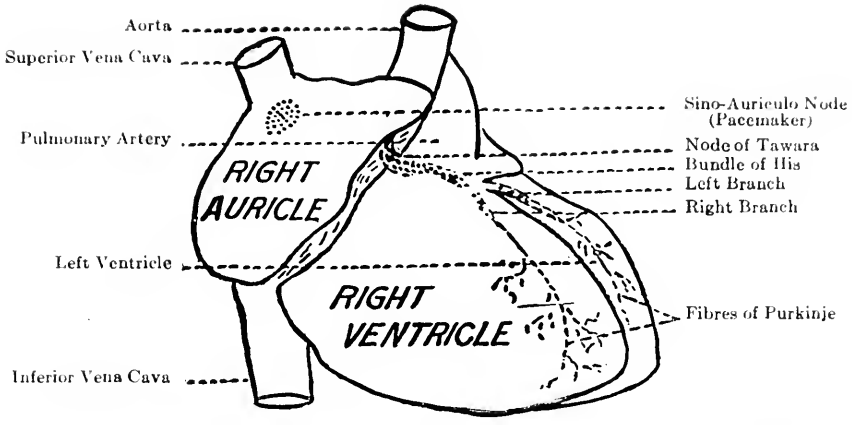


FIG. 345.—Diagram Showing the Conduction System and its Relation to the Other Anatomical Structures of the Heart.

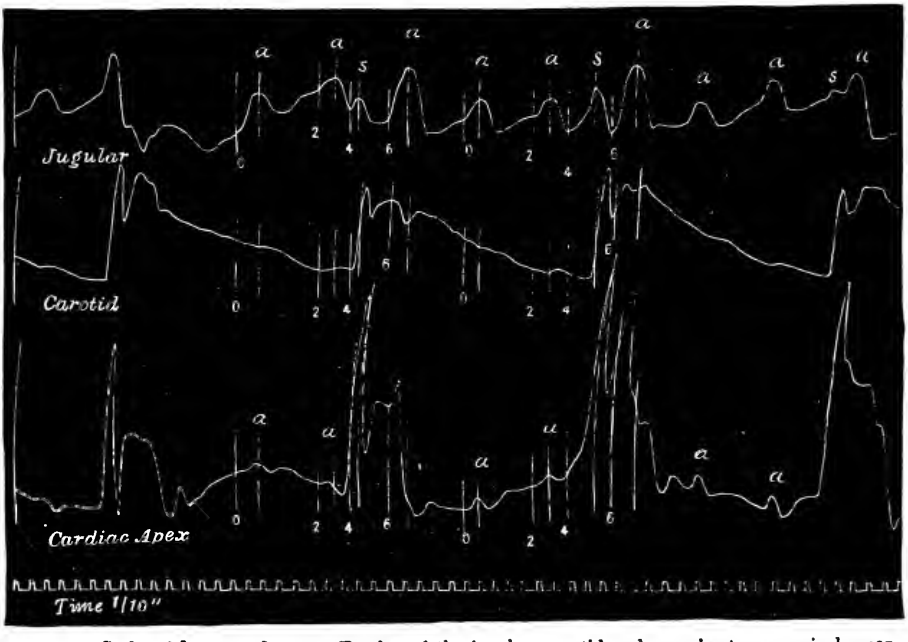


FIG. 346.—Stokes-Adams syndrome. Tracing of the jugular, carotid and apex beat. *a*, auricular contraction; *s*, ventricular contraction.—Jefferson Hospital.

Auricular Extrasystoles.—The abnormal impulse may originate in any portion of the auricle. It is transmitted to the ventricles, which take part in the premature contraction. The pause after the extra contraction is only slightly longer than the normal interval between beats.

Ventricular Extrasystoles.—The premature impulse arises from some part of the ventricles. As a rule the rhythm of the auricles is not disordered. The time between the ventricular systole preceding the extrasystole and that which follows it equals twice the interval between normal beats. There is therefore a full compensatory pause after the extrasystole. This is not the case in auricular extrasystoles.

Interpolated Extrasystoles.—When the extrasystole occurs early and the auricular rate is slow the next regular auricular stimulus may reach the ventricle after the extrasystolic refracting period. The ventricular response then occupies its normal position and the ventricular extrasystole is an extra contraction between two ventricular systoles separated by a normal interval and is designated as interpolated extrasystole.

Nodal and Retrograde Extrasystoles.—Nodal extrasystoles originate in the node of Tawara or elsewhere in the conduction system between the auricles and ventricles, the stimulus being transmitted in a retrograde direction to the auricles and in the normal direction to the ventricles with simultaneous contraction of both. When a series of ventricular extrasystoles occur in rapid succession the auricles may respond to motor impulses of ventricular origin.

In all form of extrasystole, whatever the point of origin, remarkable irregularity of recurrence may be noted. In the same individual they may at times appear only occasionally and at long intervals, while at other periods they may be frequent and more or less continuous, and these changes usually take place in the absence of obvious cause. They are sometimes scattered in a disorderly manner among the heart-beats but often appear at regular intervals of several or many beats. Again an extrasystole may follow for a time each regular systole, bringing about the modification of rhythm known as *pulsus bigeminus* or *coupled beats,* or they may succeed two normal contractions—*pulsus trigeminus.* These derangements of rhythm may, however, be encountered in heart block.

Extrasystoles are not common before puberty but are often observed in early middle life and become progressively more common as age advances. In a large proportion of the cases they are unattended by subjective secretions but very frequently the patient is conscious of the derangement of rhythm, and the pause and the stronger beat that follows it may cause much distress and anxiety, particularly at night. Their pathogenesis is not well understood. They frequently occur as a manifestation of irritability of the heart muscle in general nervous conditions, in mental overstrain and worry, under the influence of overindulgence at table or after excesses in alcohol, coffee or tobacco, in full doses of drugs of the digitalis group, after sexual excesses, in pregnancy and at the menopause. Under these conditions and very often in their absence extrasystolic arrhythmia may manifest itself during long periods of time without any other evidences whatever of cardiac trouble. Such irregularity, however, is a common element in the

symptom-complex of serious and progressive disease of the heart. These facts warrant the view that of themselves extrasystoles are of no great prognostic significance.

This disturbance of rhythm may be recognized by palpation of the radial pulse or apex beat; better by auscultation or the polygraph, but the differentiation of its various types can only be made by electrocardiography (*q. v.*).

Paroxysmal Tachycardia.—Single abnormal impulses manifest themselves as extrasystoles and arise as the case may be from the musculature of the auricles, ventricles or in the auriculo-ventricular conduction system. In the same manner multiple impulses may arise and follow each other in prolonged series lasting from a few minutes to hours or days. These ectopic rhythms are marked by an extreme rapidity, but their relationship to extrasystitus is shown by the fact that the latter forms of arrhythmia not rarely persist after the subsidence of and between the paroxysms of tachycardia.

Paroxysmal tachycardia is characterized by the great rapidity of the cardiac rhythm and the abruptness of the onset and termination of the attack. This peculiarity distinguishes this form of rapid heart from that of auricular flutter and fibrillation and that of Graves's disease. The rapid impulses usually arise from some point in the auricles. The rate varies from 120 to 200 in the minute and is generally between 140 and 180. It does not undergo changes corresponding to the erect and recumbent posture. The rate should be taken at the apex rather than at the radial. Very rarely brief paroxysms of tachycardia arise in the ventricles. The symptoms are due to the rapid rate of the heart. They consist of subjective sensations of cardiac distress and palpitation with nervousness and apprehension, and the sudden subsidence of the attack is attended with a sigh of relief. Prolonged paroxysms cause cardiac insufficiency with dilatation and stasis symptoms, breathlessness, oligæmia and œdema. The pathogenesis is practically the same as that of extrasystoles. The paroxysms occur in neurotic individuals and those suffering from sclerosis of the coronary arteries and other forms of myocardial disease. The exciting cause of the paroxysm is very often a violent emotion or sudden physical stress or unusual or prolonged mental or bodily effort. In many cases its termination follows pressure upon the vagus nerves in the neck, a series of deep respirations, eructations of gas or the act of vomiting. The prognosis is more favorable in early middle life than in the old. Frequent recurrences of the attacks with dilatation and the symptoms of cardiac insufficiency are of ominous import. Paroxysmal tachycardia may be recognized by the sudden occurrence of a very rapid pulse-rate which continues for a time and ceases as abruptly as it began, a history of previous attacks of a like character and the occurrence of extrasystoles after the heart recovers its normal rhythm and at times in the intervals between the paroxysms. The discrimination of the type as to its seat of origin—which is most commonly auricular—or ventricular or nodal, can only be made by electrocardiography.

Auricular Fibrillation; Absolute Arrhythmia; Perpetual Arrhythmia.—This disorder of rhythm is characterized by absence of the normal contraction of the auricles as a whole and persistent disorderly

contraction of the ventricles. Rapid, irregular, incoördinated contractions of the auricular muscle-bundles arise at various points and during the entire cardiac cycle. As a result of variations in the force of the impulse transmitted from the auricles and the length of the refractory periods there is no controlling rhythm as in irregularities of the extrasystolic type. The conduction of the irregular impulses from the auricles to the ventricles varies with the capacity of the auriculo-ventricular conduction system. With normal transmission the ventricular rate may be 100 or more per minute; when transmission is retarded by vagus inhibition or the action of digitalis the ventricular rate is slower, but the irregularity persists. This form of arrhythmia may occur at any period of life and is more common in males than in females. It is always associated with myocardial disease; very often with valvular lesions and is especially common in disease of the mitral valve system. It frequently follows the infectious diseases, particularly influenza, enteric fever and those due to streptococci. More than 33 per cent. of the cases give a history of acute rheumatic fever. The venous pulse gives no evidence of auricular systole. The fibrillation is fine, so that as a contracting organ the auricles are paralyzed and the concomitant symptoms are those of circulatory insufficiency. It is in accordance with these facts that in cases of mitral stenosis upon the occurrence of auricular fibrillation the presystolic murmur disappears, though a diastolic murmur, if previously present, continues to be heard. Subjective sensations of irregular action of the heart and precordial distress are common, especially upon exertion. Many of the ventricular beats do not come through to the wrist, especially when the rhythm is rapid. The ventricular rate should therefore be taken at the apex by palpation or auscultation, since there is a close correspondence between the actual frequency of the ventricular rhythm and the degree of circulatory failure. The clinical picture is characteristic and the diagnosis may be made at the bedside. The electrocardiogram is characteristic and of importance in the study of otherwise unattainable details.

Auricular Flutter.—The auricular rate is more rapid than in paroxysmal tachycardia, being from 200 to 350 per minute. The auricular contractions are regular and coördinated, causing distinct waves in the jugulars and upon the electrocardiogram. The latter shows that the pacemaker has been transposed from the sino-auricular node to a single distant point in the auricular tissue. The extremely rapid auricular rate causes a 2:1 heart block with a ventricular rate of exactly one-half—100 to 170—usually perfectly regular. Occasionally the type undergoes changes to a 3:1 or 4:1 block with corresponding alterations in ventricular rate and sometimes the degree of block undergoes rapid changes with ventricular irregularity though the auricles continue to contract regularly. Flutter is more common in elderly persons and in males. The symptoms consist of sensations of exhaustion upon slight exertion. It is of diagnostic importance that the ventricular rate is not influenced by rest, moderate exercise or change of posture. The prognosis is uncertain. Transient attacks, resembling paroxysmal tachycardia occur, but with recurrence these tend to become permanent. It sometimes happens that the heart block ceases and the ventricular rate abruptly becomes equal to that of the auricles, a change attended with loss of consciousness and the danger of sudden death. The diagnosis may some-

times be made clinically, but it is as a rule only possible by electrocardiography. Full doses of digitalis or strophanthi may convert auricular flutter into auriculo fibrillation, which may subsequently disappear with the recurrence of the normal rhythm.

Heart Block.—This term is used to designate disturbances in the conduction of motor impulses between the different chambers of the heart. Functional derangements or actual lesions of the auriculo-ventricular bundle of His may delay or prevent their normal transmission in the cardiac cycle. The degree of interference with the conduction of the impulses varies with the rapidity with which they are presented to the conducting tissue or the extent of its impairment by organic disease. Three grades are recognized: (1) delayed conduction; (2) partial heart block, and (3) complete heart block.

(1) Delayed conduction may be temporary as in severe infective diseases, as pneumonia or septic conditions, or it may mark an early stage of a progressive lesion due to rhumatism or syphilis. Clinically a provisional diagnosis may be made when there is a "reduplication of the first sound" or a "double second sound," these signs arising in consequence of the prolongation of the interval between the auricular systole and the ventricular systole. A positive diagnosis cannot be made in the absence of polygraphic or electrocardiographic aid.

(2) Partial Heart Block.—According to the degree of interference with conductivity certain auricular contractions are not passed on to the ventricles. This may happen occasionally or only every second, third or fourth impulse may be able to pass, and a definite ratio between the auricular and ventricular rhythm then arises with the production of a 2:1, 3:1 or 4:1 block as the case may be. Vagus stimulation, and large doses of digitalis and allied drugs, may cause partial block. Among the functional causes of this form of heart block must be included the exhaustion of conductivity in the His bundle by the excessive number of stimuli arising in auricular fibrillation and auricular flutter.

(3) Complete Heart Block.—Functional connection between the auricles and ventricles is wholly interrupted and the dissociation is complete. The ventricles develop an automatic rhythm which is perfectly regular and slow—30 to 40 beats per minute. This idioventricular rhythm may originate at a point in the His bundle above its division and reach both ventricles by way of the normal paths; or there may be a lesion involving the branch of the conduction system to the right or the left side permitting the stimulus to pass to the opposite ventricle only and reach the ventricle corresponding to the side of the block by way of the ventricular muscle—intraventricular block.

The Stokes-Adams Syndrome.—In complete heart block the new rhythm of the ventricles is always slower than the normal rate. Nevertheless, there are a sufficient number of regular systoles to maintain the circulation. If, however, the pulse frequency falls to a very low level, cerebral anæmia results with unconsciousness and convulsions. To this symptom-complex the term Stokes-Adams syndrome is applied. Many patients, however, who have complete heart block never have convulsions. At the time of the attack the pulse-rate is commonly about 30. It may, however, fall to 8 or 15.

Such a prolongation of the interval between ventricular systoles is attended with great danger to life. The loss of consciousness lasts a few seconds, rarely more than ten. The attacks occur singly or in groups; sometimes 30 or more occur in the course of twenty-four hours. The causes of heart block are in general (a) nervous or toxic and (b) organic. The latter may give rise to every degree of block from delayed conduction to complete dissociation. The commoner lesions involving the conduction system of His are fibroid changes, local myocarditis, abscess, gumma or new growth. Robinson found in the Museum of the Pennsylvania Hospital a heart in which a gumma is situated upon the septum in such a position as to involve this muscular bundle, and upon looking up the clinical records discovered, after the lapse of twenty-five years, that bradycardia had been a prominent symptom in the case. There is a group of cases in which no lesions are found after death. The cases due to syphilis have occurred in early adult life; those due to sclerotic processes at a later period. One of my cases, a woman, was fifty-two; another a man of seventy.

Heart block is to be distinguished from the physiological bradycardia occasionally seen in pregnancy, from that of old age, and that which occurs in the inanition of hunger. It must also be differentiated from the pathological slow pulse of convalescence from acute disease, of certain gastric affections, especially ulcer and carcinoma, of jaundice, of fatty heart, and of uræmia. Abnormal slowness of the pulse is present also in some cases of anæmia and chlorosis, and in diseases of the nervous system, as brain tumor, affections of the cervical cord, and apoplexy. Certain poisons, as opium, alcohol, lead, and digitalis, cause bradycardia. The differential diagnosis between heart block and any of these conditions rests upon the essential difference between the frequency of the auricular contractions as manifest in the jugular pulsation and those of the ventricles as felt in the radials and at the apex and the exclusion of other causes. In many cases of cardiac insufficiency a considerable proportion of the ventricular systoles fail to transmit a pulse-wave to the wrist. The apex beats must be counted in all cases of slow pulse.

A provisional diagnosis of heart block may be made when there are dropped beats or when there is a continuous marked bradycardia, especially when associated with a history of attacks of unconsciousness (Stokes-Adams syndrome); but the polygraph or electrocardiograph are necessary to the making of a positive diagnosis and its type.

IV. THE ORGANIC DISEASES OF THE HEART.

Under this heading are considered diseases of the myocardium, the pericardium and the endocardium, chronic valvular diseases and congenital defects of the heart.

I. DISEASES OF THE MYOCARDIUM.

i. Acute Myocarditis.

Definition.—Acute inflammation of the wall of the heart. Pathologically parenchymatous and interstitial forms are recognized. There is cell infiltration of the interstitial tissue, associated with granular, fatty,

and hyaline degeneration of the muscle fibres, and loss of their contractile elements. There are three varieties: 1. Primary Acute Myocarditis.—In the absence of any recognized cause, or after a wetting, the symptoms of an acute interstitial inflammation of the myocardium develop with irregular fever and great weakness, and after a course of several days or a week or two terminate in death. Primary acute myocarditis is an exceedingly rare affection, the occurrence of which as a separate disease has been questioned. 2. Secondary Acute Myocarditis.—This variety occurs in the course of or during the convalescence from many of the specific infectious diseases. 3. Acute Septic Myocarditis.—In a majority of the cases infection of the myocardium takes place by way of the coronary arteries. There are commonly multiple miliary abscesses. In other instances abscess formation follows the direct invasion of the heart muscles from the valvular or mural lesions of a septic endocarditis.

Etiology.—Predisposing Influences.—These are wholly unknown. No plausible explanation of the fact that the myocardium suffers in some cases of infectious disease and sepsis, and escapes in others, has yet been adduced. There is nothing in age, sex, or previous health to account for this difference. Acute secondary myocarditis occurs in diphtheria, enteric and typhus fevers, scarlet fever, rheumatic fever, variola, and gonorrhœal infection. It is especially frequent in association with diphtheria. The septic form is less common.

Exciting Cause.—The primary form has followed cold and exposure and traumatism to the left chest. The local action of specific toxins, the arrest of minute infected emboli in the branches of the coronary arteries, and direct bacterial invasion from the endocardium are the immediate causes of the myocardial inflammation.

Symptoms.—The clinical manifestations are usually obscured by those of the primary affection. Profound cardiac asthenia manifests itself by feeble heart sounds, a scarcely perceptible radial pulse, arrhythmia, and acute dilatation. Sudden increase of pallor, apathy, somnolence, and precordial distress occur. Vomiting is common and sometimes urgent. In some cases there is restlessness and agitation; in others subjective sensations are absent and the gravity of the complication reveals itself only by the objective manifestations.

Diagnosis.—Direct.—The diagnosis must, in a majority of the cases, be a provisional one. Only in the light of later events, often at a remote period in cases that have recovered, can the cause of the grave cardiac symptoms during the acute attack be recognized. The symptoms of chronic myocardial changes in an individual who has passed through a grave attack of one of the febrile infections are significant.

Differential.—The intensity of the process varies. The average cases are to be differentiated from the acute cardiac asthenia of influenza by the rapidity with which the latter develops and the peculiar psychic manifestations with which it is so frequently associated; from acute cardiac exhaustion from overwork or athletic excesses by the history of the case; and from all of these by their occurrence in the absence of especial exposure to wet and cold, or of an acute infection, or of sepsis.

Prognosis.—The outlook depends largely upon that of the primary disease, to which the complication adds additional gravity. In diphtheria it is extremely grave; in gonorrhœal infection scarcely less so. In rheumatic fever it is more favorable, and in that form which occurs in the convalescence from enteric fever and scarlatina recovery is the rule. The prognosis in the septic cases is unfavorable. The danger of sudden death, even in cases in which the symptoms are of moderate grade, is always to be considered. It is probable that the sudden death which occasionally occurs in the late convalescence of croupous pneumonia is due to acute myocarditis.

ii. Chronic Myocarditis.

Chronic Insufficiency of the Heart Muscle; Myodegeneratio Cordis.

Definition.—Chronic inflammation of the heart muscle, characterized anatomically by round-celled infiltration of the interstitial connective tissue, followed by fibrosis and parenchymatous degeneration of the muscular fibres, and clinically by impairment of the function of the heart.

Etiology.—Predisposing Influences.—Chronic myocarditis in its simplest form is a late manifestation of acute myocarditis in cases that survive. From this point of view the acute infections constitute predisposing influences of great importance. It is especially common after rheumatic fever, occurring occasionally in cases without valvular disease, and being, to some extent at least, a constant accompaniment of chronic valvular disease. It often has its beginning in extensive pericardial adhesions. It may follow diphtheria, enteric fever, variola, scarlet fever, gonorrhœa, and the septic cases in which recovery as to the general condition takes place. Chronic myocarditis frequently has its origin in the lesions of syphilis. It is met with in chronic alcoholism and in saturnine intoxication. Arteriosclerosis, gout, and chronic renal disease especially predispose to this form of myocarditis. Habitual overstrain of the heart, such as occurs in athletes and those who follow laborious occupations, as stevedores, iron workers, coal passers, and the like, is a predisposing influence of great importance; nor can we overlook the part played by prolonged mental stress and depressing emotions. More than 90 per cent. of cases of heart disease involving the heart muscle chiefly or the valves and myocardium in common are due to rheumatism (streptococcic infection), syphilis, arteriosclerosis or renal disease.

In the great majority of the cases the symptoms first appear after the fortieth year. They may occur at a much earlier period. The insufficiency of the heart following the acute infections with or without valvular disease is due to myocardial lesions. The average age of first manifestations in the adult is later in women than in men. Men suffer more commonly than women, a fact in harmony with the different modes of living in the sexes in early and middle adult life. Several active predisposing influences are frequently present in the same case.

Exciting Cause.—The immediate causes are, (1) a disproportion between the power of the heart and the work which it is called upon to perform, (2) infectious, and (3) toxic. These may be active in various

combinations. It is not always possible to determine the actual cause or even to ascertain that which predominates.

Symptoms.—The clinical picture is a variable one. Its fundamental characters depend upon a single pathological condition, namely, cardiac inadequacy. The want of power varies in degree. It may be slight and only manifest upon unusual effort, or so great that the circulation fails to meet the ordinary needs of the organism at rest. An early and an advanced stage have been described. This division is misleading, since the disease is not always progressive, and there are cases in which the early stage is absent or the advance of the disease so rapid that the beginnings are not recognized, and cases in which periods of improvement occur, and, finally, a small proportion of cases in which, after a time, an actual recovery takes place. Furthermore, the range of symptoms varies according to the anatomical and physiological peculiarities of the individual, the integrity of his organs, and condition of his nervous system.

In the beginning the attention of the patient is aroused by subjective symptoms, as palpitation, precordial distress, or pain and oppression induced by physical or mental effort. In another group of cases, after repeated mild attacks of angina or a single severe attack, the general symptoms of cardiac insufficiency develop. Moderate effort, the daily work, mounting stairs, climbing a gentle ascent, a generous meal, cause oppression and shortness of breath. Presently in many cases a moderate dyspnœa persists. Epigastric weight, loss of appetite, constipation or diarrhœa, and slight pretibial œdema mark an advance in the progress of the malady. Loss of strength, anæmia, emaciation, dizziness, and syncopal attacks may now occur. The grave symptoms consist of continuous dyspnœa of greater intensity and interrupted by asthmatic attacks, icterus, oliguria, albuminuria, increasing œdema, and effusions into the serous sacs. The pallor becomes more marked, and cyanosis varying in degree appears around the lips and at the finger-tips. The nervous symptoms are intensified. Palpitation, precordial pain, and oppression may be continuous or recur in alarming paroxysms. Complications are frequent and in many cases terminal. The more common are bronchitis and bronchopneumonia. Less frequent are inflammations of the serous membranes, pleurisy, pericarditis, and peritonitis. At any period in the course of the disease blood-stained sputa may be noted. In the chronic passive congestion of the lungs which accompanies advanced heart disease a reddish sputum is common, the color being due to great numbers of cells containing pigment granules—"heart-failure cells."

Physical Signs.—Early in the disease the signs of moderate dilatation of the left ventricle and, to a less extent, of the right ventricle are apparent. In many of the cases, however, and especially in those in which the signs of valvular disease are absent, and those characterized by angina pectoris, the heart is not enlarged. With improvement in the general symptoms, the early dilatation of the heart usually disappears. The sounds may be for a long time normal. In some cases the first sound is accompanied by a faint inconstant murmur and the second pulmonary is accentuated; or again a mitral systolic murmur, sometimes indistinct and soft, sometimes loud and well marked, accompanies or replaces the first sound—the murmur of muscular mitral insufficiency. This murmur sometimes has a musical

quality. Other murmurs are not common in the milder cases of myocarditis. In the graver cases dilatation to the left and upward, and usually toward the right, is almost constant. The transverse dulness is increased and the apex beat displaced to the left and slightly upward. The enlargement of the heart frequently increases and diminishes in accordance with the varying intensity of the symptoms, but a return of the heart to its normal size is no longer to be hoped for. Upon auscultation the increased muscular insufficiency is manifest in a well-characterized mitral systolic murmur, which after a time is associated with a tricuspid systolic murmur. Very rarely a faint aortic diastolic murmur may be recognized. These murmurs in many cases undergo rapid changes, corresponding to the degree of dilatation with the gain or loss in the power of the heart, and sometimes wholly disappear. In other cases they are persistent, and, when associated with hypertrophy, as in chronic nephritis, they cannot always be differentiated from the murmurs of organic valvular disease.

The pulse is usually weak and small. Sometimes, especially in the form of chronic myocarditis associated with interstitial nephritis, it shows increased tension. Its frequency is much increased by moderate exertion. In a majority of the milder cases arrhythmia does not occur; exceptionally it is an early sign. In a small proportion of the cases the pulse is slow. As the insufficiency becomes more marked the pulse-frequency augments and arrhythmia appears. The latter is often of high grade, both as to the time of the beats and as to their force. The inequality of the ventricular contractions is such that many of them fail to transmit the pulse-wave to the radials, so that the pulse counted at the wrist is less frequent than the impulse counted at the apex.

The following forms demand separate consideration: The Inflammatory Form.—The symptoms are those already described. The affection occurs as a later stage of acute myocarditis, showing itself in some instances during the convalescence from the primary disease; in others not until a remote period. There are cases in which recovery takes place. In this connection it is to be noted that fibroid degeneration is sometimes the reparative process by which destructive lesions are corrected. The Form Due to Diseases of the Coronary Arteries.—*Sclerosis of the Coronary Arteries.*—The lesion consists chiefly of a thickening of the intima. It is either diffuse or circumscribed, and leads to narrowing of the lumen of the affected vascular twigs. The left coronary artery and its branches are involved much more commonly and to a greater extent than the right. When the narrowing of the lumen is of high grade, or there is complete occlusion of the affected vessels, infarction of the heart muscle—*myomalacia cordis* (Ziegler)—occurs. The necrotic focus is invaded by connective-tissue elements and converted into a fibroid cicatrix. There may be a diffuse increase of the interstitial tissue. This variety of chronic myocarditis presents symptoms of muscular insufficiency, but is especially characterized by a tendency to angina pectoris and so-called cardiac asthma, a special liability to precordial pain, and the signs of narrowing of the aortic orifice. In fact, arteriosclerosis of the coronary arteries is very commonly associated with similar pathological changes in the aortic valves and the beginning of the vessel itself. A systolic aortic murmur, usually

not so loud and coarse nor beginning so promptly with the systole as in pure valvular stenosis, and followed by a well-marked, even accentuated, second sound, is usually heard. Dulness to the right of the sternum in the second and third interspaces and jugular pulsation are associated signs. *Embolism and Thrombosis of the Coronary Arteries.*—These lesions cause myocardial infarct. Thrombosis may result from sclerosis; embolism from various forms of infection, or very rarely from fragments of atheromatous plates. Death directly follows the closure of the coronary artery at its origin. Elsewhere the occlusion of the artery is followed by infarction and death after several days or, in rare cases in which the extent of the softening is limited, by recovery. The local necrosis may lead to rupture of the heart. The changes in the heart muscle depend upon the extent and degree of the sclerosis and the rapidity with which the blood supply to the muscle is arrested.

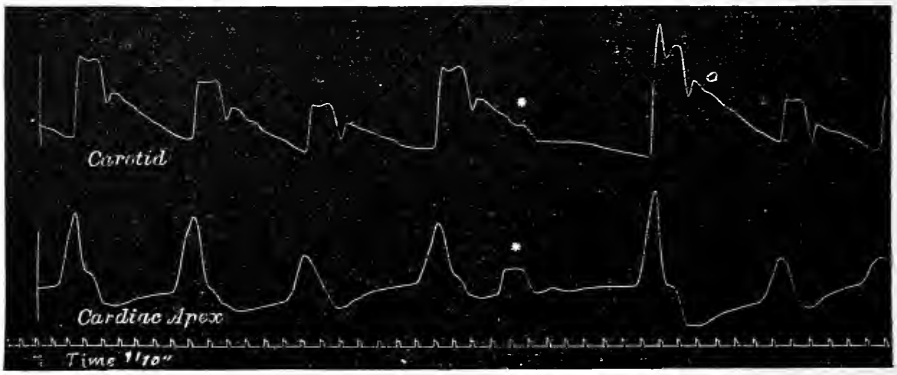


FIG. 347.—Tracing of the carotid artery and cardiac apex in chronic myocarditis showing irregularity in time and in force. * Ventricular contraction not of sufficient strength to produce a distinct carotid wave.

GENERAL ARTERIOSCLEROSIS IN CHRONIC MYOCARDITIS. — Habitual increase in the arterial blood-pressure is an important cause of chronic myocarditis. Prolonged muscular exertion, toxic substances, and arteriosclerosis alone or associated with chronic disease of the kidneys are potent factors. The changes in the myocardium develop gradually, and the symptoms and course of the disease are not different from those of the ordinary form. Middle-aged men who have lived well suffer from this form and the early symptoms in such cases are usually abdominal—fulness, weight, flatulence, and constipation.

SYPHILIS OF THE HEART.—Single or multiple gummata are very rare. Diffuse interstitial myocarditis is more common. Syphilis of the heart may be congenital or acquired. In the acquired form it is usually a tertiary manifestation. The condition is very often latent, but when well developed it corresponds clinically to the ordinary form of chronic myocarditis.

NUTRITIONAL DISORDERS AND CHRONIC MYOCARDITIS. — The myocardium suffers in the same way as the skeletal muscles. Previously existing myocarditis constitutes an important predisposing influence. The cardiac insufficiency may show itself in the morning when the duties of the day are undertaken after an insufficient breakfast. Free purgation

may cause symptoms. A too strict antidiabetic diet and the injudicious treatment of obesity are to be considered. This form of cardiac inadequacy is common in the gouty, partly because of anomalous metabolism, partly because of insufficient food, and often as the result of an unwise use of colchicum or other drugs. To this category must also be referred the feeble heart of anæmia and chlorosis, together with the atrophy of the heart in the acute febrile infections, tuberculosis, and starvation. Long recumbency, as in illness or those who from other causes are bed-ridden, leads to gradual heart starvation. The symptoms are those of cardiac dilatation, dyspnœa upon exertion, gastrohepatic distress, œdema of the lower extremities, and general asthenia.

Diagnosis.—Direct.—Objective phenomena dependent upon changes in the heart upon which to base a diagnosis are usually absent. The anamnesis and the cardinal condition of weakness of the heart, as manifest by rational symptoms, justify a provisional diagnosis. A positive diagnosis may be reached by the method of exclusion. In a considerable proportion it cannot be reached at all.

In the anamnesis the age of the patient, his mode of life, the history of antecedent disease are important. The present condition of the superficial arteries, the blood-pressure, the ophthalmoscopic evidences of changes in the walls of the retinal arteries, the specific gravity of the urine and the presence or absence of albumin, tophaceous deposits in the ears or around the small joints, and the occurrence of asthma-like seizures, precordial pain, and angina pectoris are all to be carefully investigated. Dyspnœa, precordial and abdominal distress, diminution in the urine, and œdema are most significant symptoms; feebleness of the heart sounds, a weak or undiscoverable impulse, a faint or inconstant mitral systolic murmur are signs of importance. A marked increase in the pulse-frequency upon slight exertion is very suggestive.

The diagnosis of sclerosis of the coronary arteries may be made when, in a person of middle age presenting the signs of sclerosis of the aortic valves or atheroma of the ascending aorta, attacks of paroxysmal dyspnœa or angina pectoris occur in connection with the manifestations of cardiac inadequacy. The diagnosis of coronary embolism and thrombosis is usually impossible. These conditions frequently arise in advanced cases without any clinical manifestation whatever. The evidences of general arteriosclerosis in a case characterized by the signs of great and persistent cardiac weakness are of diagnostic importance. The symptoms of chronic nephritis, and, in particular, of contracted kidney, are also significant. Finally, transient attacks of increased cardiac asthenia in persons improperly nourished, overworked, much torn by the stress of life, or suffering from acute or chronic disease, are very often the manifestations of heart starvation.

Differential.—*The Cardiac Neuroses.*—The differential diagnosis is rendered uncertain, especially in the early cases of chronic myocarditis, by the fact that the murmurs and especially neurasthenic symptoms are very common in myocarditis. A history of accidental or surgical trauma, overwork, anxiety, grief, onanism, sexual excess, hysteria, speak for a cardiac neurosis. The age of the patient is of some value in diagnosis. Under forty the neuroses are more common; after forty myocarditis.

Chronic Valvular Disease.—Here also the frequent association of the two conditions gives rise to nice problems in differential diagnosis. The difficulty relates usually to mitral insufficiency. The question concerns the nature of the cardiac insufficiency. Is it valvular or muscular? A systolic murmur, accentuation of the second pulmonary sound, and cardiac dilatation occur in both conditions. Hypertrophy of the left ventricle and a strong impulse are in favor of valvular disease. These conditions may be absent in moderate mitral insufficiency, or the hypertrophy may be due to renal disease or adherent pericardium, or again the impulse may be obscured in emphysema. Under these circumstances a history of rheumatic fever, or the infrequency of myocarditis in childhood, or of uncombined mitral insufficiency in the aged is to be considered. Furthermore the murmur of muscular insufficiency is changeable and may wholly disappear with improvement of the condition of the myocardium under rest and treatment. In fact the outcome of a reasonable management of the case is of diagnostic value, since interstitial changes and parenchymatous degeneration are usually progressive and ultimately give rise to symptoms that cannot be misunderstood.

Prognosis.—Chronic myocarditis can scarcely be said to end in recovery. Circumscribed lesions may be repaired by the development of fibroid tissue. As a rule the outlook is absolutely unfavorable. Much depends upon the circumstances of the patient and his ability to bring himself under discipline. The sooner this is done and the slighter the lesions, the better the outlook. Sometimes improvement and the postponement of the lethal disaster occur in the most alarming cases.

iii. Hypertrophy and Dilatation.

(a) HYPERTROPHY OF THE HEART.

Definition. — An anatomical condition characterized by an overgrowth of the myocardium. A single chamber may be involved, or one side, or the whole heart. The left ventricle is most commonly affected.

The wall may be thickened without enlargement of the chamber, or the chamber may be dilated,—*eccentric hypertrophy; combined hypertrophy and dilatation,*—and this is by far the most common form. Thickening of the walls with diminished capacity of the chambers may be simulated by post-mortem conditions—so-called *concentric hypertrophy.*

Etiology.—The work of the heart is increased but the nutrition of its wall is maintained. (a) In its simplest form hypertrophy is caused by habitual excessive muscular exertion. It is not possible to say when physiological increase passes over to actual hypertrophy. The heart of the iron worker is much larger than that of the clerk. Muscular work increases blood-pressure. General arteriosclerosis, particularly that form associated with chronic nephritis, toxic substances, as lead, alcohol, and the poisons of gout, increases the arterial tension and is a common cause of hypertrophy. (b) The freedom of the movements of the heart is hampered by extensive pericardial adhesions, by local or general interstitial myocarditis, and by the neuroses which give rise to cardiac overaction, especially exophthalmic goitre, tachycardia, and those resulting from the abuse of various stimulants

and narcotics, especially tea and coffee. The heart works at a disadvantage and if its nutrition remains fairly good undergoes hypertrophy. (c) The amount of blood which the heart has to propel is increased in aortic and mitral insufficiency, since with every revolution of the heart a given quantity of blood passes to and fro through the affected orifice like a shuttlecock and demands a proportionate increase in cardiac power to maintain the equilibrium of the circulation. Habitual excesses at table and, in particular, the consumption of enormous quantities of beer, have been shown to give rise to cardiac hypertrophy. (d) Resistance to the free outflow of blood caused by narrowing of an orifice causes hypertrophy of the wall of the affected chamber, as the left ventricle in aortic stenosis, the left auricle in mitral stenosis and lesions of the pulmonary valve. Congenital narrowing of the aorta or of the arterial system in general is another cause of hypertrophy.

The various conditions which primarily or chiefly cause hypertrophy of the left ventricle ultimately cause also hypertrophy of the right ventricle. Hypertrophy of the right ventricle arises also in other conditions which increase the resistance in the pulmonary vessels, such as emphysema, fibrosis of the lungs, and deformities of the chest. The left auricle undergoes dilatation with hypertrophy in mitral disease, especially stenosis; the right in conditions characterized by increased blood-pressure in the pulmonary circuit, both of valvular and of pulmonary origin.

Enormous enlargement of the heart—*cor bovinum*—is usually due to aortic insufficiency, chronic mediastinitis, or chronic interstitial nephritis.

Hypertrophy of the heart is essentially a conservative process. Its development is gradual and for a time keeps pace with the advance of the lesions with which it is associated. So long as the equilibrium of the circulation is maintained, the hypertrophy is *compensatory;* when the hypertrophy begins to fail, the compensation is *impaired;* when dilatation is marked and cardiac insufficiency is extreme, the compensation is said to be *ruptured* or *broken.*

Moderate hypertrophy is unattended by marked direct symptoms. In fact it prevents to a great extent the manifestations of the underlying disease. Sensations of fulness, aggravated when lying upon the left side, easily induced overaction, and the consciousness of precordial pulsation or throbbing in the neck or head sometimes occur in advanced cases.

The physical signs depend upon the degree of hypertrophy and its preponderance over the accompanying dilatation, and the extent to which the heart is covered by the overlying borders of the lungs. When the hypertrophy is advanced and dilatation yet subordinated to it, the diagnosis may be readily made. Upon inspection the impulse is strong and extended to the left and downward. In marked enlargement it is heaving, and the whole precordial area may pulsate. There is pulsation at the root of the neck. A visible pulsation in the epigastrium occurs in hypertrophy of the right ventricle. Palpation confirms the signs obtained by inspection, and in women with large mammæ may enable the examiner to ascertain the position of the apex when inspection and percussion are ineffectual. The radial pulse is full, strong, and rather slow. Upon percussion the areas of relative and absolute dulness are usually both increased; in large-

lunged persons and cases of emphysema, the relative dulness only. Extension of deep dulness to the left and upward is a sign of hypertrophy of the left ventricle; to the right and downward a sign of hypertrophy of the right ventricle. Upon auscultation the first sound is loud, often booming and prolonged; the second aortic sound accentuated in left ventricle hypertrophy, the second pulmonary accentuated when the right ventricle is involved. Accentuation of the second aortic occurs, however, in those conditions of high tension of the systemic arteries which cause left ventricle hypertrophy, and accentuation of the second pulmonary in those which interfere with the normal flow of blood through the pulmonary vessels.

(b) DILATATION OF THE HEART.

Definition.—Dilatation of the heart is an anatomical condition, characterized by an increase in the size of its chambers due to stretching of its walls. Dilatation may affect one or more chambers of the heart. It may be acute or chronic. Acute dilatation is usually primary; chronic dilatation secondary to cardiac insufficiency or valvular disease. A previously diseased heart is more liable to acute dilatation than a normal heart.

The capacity of the chambers of the heart varies considerably within normal limits. Pathological enlargement—*dilatation*—exists when the affected chamber is unable to empty itself of blood in systole, and is permanent. The myocardium, like other muscles, increases within certain limits with use, especially if the increase in work is gradual and nutrition is maintained. Dilatation accompanies failure of tonicity.

Etiology.—Dilatation results from a disproportion between the power of the heart muscle and the work which it has to do. In other words, the pressure within the chambers may be increased or the muscular wall may be weakened. These factors may act singly or in combination. Increased pressure arises when there is an abnormal quantity of blood to be propelled or an abnormal resistance to be overcome. Under certain conditions, as in the gradually developing sclerotic form of aortic stenosis, and in the cardiovascular changes of chronic nephritis, the left ventricle may not undergo dilatation but hypertrophy.

Severe and prolonged muscular effort is a common cause of acute dilatation, as in mountain climbing or prolonged tests of endurance. The condition is known as heart strain. The symptoms are breathlessness upon exertion, cyanosis, lassitude, mental depression, and swelling of the ankles; the signs feebleness of the cardiac impulse, small, rapid, irregular and intermittent pulse, faint heart sounds, and upon percussion the evidences of enlargement of both the superficial and deep areas of dulness, increase in the area of liver dulness, and hypostatic congestion of the lungs. Relative insufficiency is shown by the development of a mitral systolic murmur, and the safety valve function of the tricuspid by a systolic murmur at the lower border of the sternum to the right. Under appropriate treatment, into which rest in the recumbent posture largely enters, gradual recovery takes place with disappearance of the signs of dilatation. In many cases they recur upon further unusual effort, and in some the damage to the wall of the heart is permanent. Acute dilatation occurs also in

consequence of undue effort after acute illness, in Graves's disease, exophthalmic goitre, and paroxysmal tachycardia. It may follow any unusual effort in a case of chronic myocarditis.

The ultimate tendency of chronic myocarditis is to dilatation. In many forms the increase in the size of the chambers and stretching of the walls begin early; in others not until after an initial hypertrophy. The symptoms are those of cardiac insufficiency; the signs those of enlargement of the heart, displacement of the apex to the left and downward, feeble, undulating impulse extending over several intercostal spaces, and faint cardiac sounds reverting to the fetal type. Dropsy and the evidences of visceral congestion are present in well-marked cases.

Valvular disease is a constant cause of dilatation of the heart. In stenosis a portion of the normal quantity of blood that should pass the affected orifice is held back in systole; in incompetency a portion of the quantity that has passed the orifice flows back in diastole. In one case the blood entering the chamber meets blood that should have passed on; in the other blood enters the chamber in diastole from two opposite directions, one physiological, the other pathological. The result is dilatation of the affected chamber and transference of the increased blood-pressure backward from the site of the valvular lesion, from ventricle to auricle on the left side, through the pulmonary circuit, to ventricle and then to auricle on the right.

Hypertrophy at first, then dilatation of the left ventricle thus results from aortic stenosis and from aortic insufficiency; hypertrophy and dilatation of the left auricle from mitral stenosis and mitral insufficiency; pulmonary hyperæmia from either stenosis or insufficiency of the aortic or mitral valve systems; hypertrophy and dilatation of the right ventricle from pulmonary hyperæmia due to valvular disease of the left side of the heart, or to intrapulmonary conditions which increase the resistance in the pulmonary circuit; hypertrophy and dilatation of the right auricle from overdistention of the right ventricle.

Whenever dilatation is in excess of hypertrophy there is a tendency to the transference of blood-pressure from the arterial to the venous side of the circulation.

When the myocardium is weakened by, (a) the toxins of the acute infections, (b) the extension of the inflammation in endocarditis or pericarditis, (c) the malnutrition of starvation in any form, anæmia, or chlorosis, dilatation may result in the absence of increase in the blood-pressure.

iv. Fatty Heart.

Fatty Overgrowth; Fatty Infiltration; Cardiac Inadequacy of the Obese; Cor Adiposum.

Definition. — A condition common in fat persons, characterized anatomically by excess of fat beneath the epicardium and among the strands of muscular fibres, and clinically by cardiac insufficiency.

It has been customary to include, under the designation fatty heart, fatty degeneration of the heart muscle and fat overgrowth or infiltration.

The present tendency is to restrict the term to the peculiar changes that occur in corpulent persons, since fatty degeneration is a common form of parenchymatous degeneration in myocarditis due to many different causes, and wholly without distinctive etiological or clinical features. Fatty degeneration of the heart occurs in the infectious fevers, wasting diseases, and the cachexias. It is very marked in acute yellow atrophy of the liver, phosphorus poisoning, and pernicious anæmia. It constitutes one of the most important changes of old age. The degeneration may also under all these conditions affect the various viscera. The wall of the heart may be locally or generally involved. The heart muscle is flabby, relaxed, and friable. The heart when thrown upon the table sinks into a shapeless mass. Microscopically the fibres are filled with minute fat particles.

The condition under consideration is entirely different. It is essentially an affection of the corpulent. There is an excess of the subpericardial fat, so great in some instances as to wholly envelop the muscle in a casing of fat. It is usually more abundant in the intraventricular grooves, along the course of the coronary arteries, and upon the wall of the right ventricle. It penetrates the muscles, separating the fibres, and may extend to the endocardium. The heart is dilated and its wall flabby and relaxed. Upon microscopical examination the muscular fibres are found to be atrophied and in some instances to have undergone fatty degeneration. There is a disproportion between the size of the heart and the requirements of the body. In many fat persons with well-developed muscles cardiac insufficiency does not occur. Cardiac symptoms are marked in that type of obesity characterized by anæmia, flabby muscles, and indolence.

Etiology.—The causes of fatty infiltration of the heart are those of the obesity of which it constitutes such an important part. The middle periods of life, the male sex, heredity, addiction to the pleasures of the table, much fluid, malt liquors and alcohol in general, luxurious habits, and indolence are potent factors in the production of corpulence and the fatty heart. Disturbances of the endocrine glands, especially the gonads and the pituitary, are important factors.

Symptoms.—The clinical manifestations are those of cardiac inadequacy, dyspnœa, a feeble pulse, much accelerated upon effort and commonly intermittent and irregular. Poor appetite, much thirst, and constipation are common. Such patients are often drowsy by day and sleepless by night. Dropsy of the lower extremities is sometimes marked.

Physical Signs.—The methods of physical examination usually yield unsatisfactory results by reason of the excess of subcutaneous fat. This is particularly true of inspection, palpation, and percussion. The results of auscultation are sometimes more satisfactory. The heart sounds are usually feeble and distant. When, however, they are well defined and the aortic sound distinct, they may be regarded as indicating fairly well-maintained myocardial nutrition. When, on the other hand, the first sound is extremely faint or replaced by a systolic murmur, the aortic sound feeble, and the pulmonic sound accentuated though feeble, the integrity of the heart muscle is greatly impaired.

Diagnosis.—The recognition of fatty heart depends upon the association of cardiac insufficiency with obesity.

The DIFFERENTIAL DIAGNOSIS between this condition and the primary anæmias, especially chlorosis and pernicious anæmia, may be made by a proper blood examination. The blood of corpulent persons of the pallid type who suffer from fatty heart may show the characters of a more or less marked secondary anæmia, but not, in the absence of specific lesions, the characters of the primary anæmias.

Prognosis.—The outlook in general is unfavorable and is rendered more so by unwise attempts to reduce the weight of the body by insufficient food, unduly increased exercise, exhausting baths, or depressing drugs. Thickened arteries, paroxysmal dyspnœa, and angina pectoris are of ominous significance.

v. Various Degenerations, New Growths, and Parasites of the Heart.

Degenerations of the heart muscle not already considered are amyloid degeneration, the hyaline transformation of Zenker, and calcareous infiltration. None of these is recognizable during life nor of clinical interest.

Tumors of the heart are commonly carcinoma and sarcoma. They are usually secondary. Fibroma, lipoma, myoma, gumma, and leukæmic infiltrations are extremely rare. Malignant tumors very commonly lead to pericarditis, which may be plastic or purulent.

Of the parasites which affect the myocardium, the echinococcus is the most common. It selects the right ventricle twice as often as the left. So long as it remains within the myocardium it does not occasion symptoms. When it finds its way into the interior of the heart it gives rise to embolism, especially in the lungs. The cysticercus and trichinella find access to the heart muscle, but do not occasion symptoms.

vi. Wounds and Foreign Bodies.

External injuries, as stabs and gunshot wounds, are very common. Their diagnosis is obvious. The subject belongs to surgery, and has acquired great importance in consequence of the recent success which, in stab wounds, has attended the prompt exposure of the heart and suturing of the wound. Internal injuries are extremely rare. They are caused by foreign bodies—a bone or artificial denture ulcerating its way from the œsophagus, or in the case of insane or hysterical persons by pins or needles that have been swallowed. In the former instance the nature of the lesion would be recognized by the history of the case and sudden fatal hæmatemesis, in the latter pericarditis would occur; but a positive etiological diagnosis *intra vitam* cannot be made.

vii. Rupture of the Heart.

This accident may occur as the result of the arrest of the blood supply to the affected area in consequence of sclerosis or embolism of a branch of a coronary artery, inducing acute softening—*myomalacia cordis*. The

heart wall may undergo similar circumscribed impairment from suppurative myocarditis or a softening gumma. Local fatty degeneration is the most common cause. The rupture occurs most frequently on the anterior wall of the left ventricle near the septum. The softened area gradually yields, and upon some effort which causes heightened intraventricular pressure, as ascending a staircase or straining at stool, it suddenly gives way and the escape of blood into the pericardial sac—*hæmopericardium*—is followed by death. Rupture in the posterior wall of the left ventricle is much less common, and rupture of the wall of the right ventricle or the auricles very rare.

Traumatic rupture of the heart may result from violent blows or contusions of the thorax, such as occur in falls or railroad accidents. This variety of heart rupture is more apt to involve the right ventricle or an auricle. In some cases the borders of the rent maintain their position. Pericardial adhesions may occur and death may be postponed for several hours or days.

Diagnosis.—In the majority of the cases death results at once and the diagnosis is impossible. When the opening is small, signs of internal hemorrhage—feeble pulse, oppression, air hunger, ghastly pallor, and orthopnœa—are suggestive. When pericardial adhesions exist the blood outflow is hindered and life may be correspondingly prolonged.

Prognosis.—The outlook, however, is without hope.

viii. Aneurism of the Heart.

Aneurism of a valve may result from malignant endocarditis. The condition is not common. The aortic valves are affected with greater frequency than the mitral. The cusp shows a bulging in the direction of the ventricle, which presently ruptures, causing acute insufficiency. The signs are not characteristic and are obscured by the primary changes. A positive diagnosis cannot be made.

Aneurism of the wall is also a rare condition. Its most common position is the left ventricle in the region of the apex, which is the portion of the wall of the heart most commonly affected in the fibroid degeneration of chronic myocarditis.

Etiology.—This condition mostly follows chronic myocarditis, but has been observed in acute mural endocarditis. Wounds of the heart and gumma are also etiological factors. The dilatation is usually single, but may be multiple.

Diagnosis.—Direct.—The symptoms are not characteristic. The associated myocarditis causes cardiac inadequacy, manifest by the usual clinical phenomena. When the tumor attains considerable size it is usually lined by laminated clots and may give rise to irregular enlargement of the diameters of deep cardiac dulness. In other cases there may be bulging in the region of the apex and perforation of the wall of the chest.

The differential diagnosis from mediastinal or pleural tumor is to be considered. The feeble pulse of cardiac inadequacy may be in marked contrast to the cardiac impulse. The X-rays may be of service in the differential diagnosis.

ix. Atrophy of the Heart.

Definition.—A diminution of the heart in weight and size. A single chamber or the entire heart may be atrophied.

The term hypoplasia of the heart is used to designate congenital undersize.

The myocardium is of a dark, reddish-brown color, and abnormally resistant. The surface is often marked or puckered. The muscle fibres are diminished in size, their transverse striæ indistinct and presenting collections of yellowish-brown pigment near the nuclei.

Etiology.—The small size of the left ventricle in extreme mitral stenosis may be looked upon as an example of atrophy of a single chamber of the heart. Common causes are starvation and wasting diseases, as cancer, diabetes, protracted suppuration, and, in particular, phthisis. Brown atrophy of the heart is common in advanced valvular disease and old age—*the senile heart.*

Symptoms.—The symptoms are those of cardiac inadequacy—feeble and rapid action, especially upon exertion, weak and irregular pulse, faint sounds, and indistinct impulse. The shrunken lungs usually increase the area of superficial dulness, but the diameters of deep dulness are reduced.

Diagnosis.—The calcification of the costal cartilages in the aged often renders the examination of the heart by percussion very difficult and unsatisfactory. The X-ray examination yields more definite signs of a reduction in the size of the organ. The clinical phenomena are much subordinated to those of the primary affection.

Prognosis.—The outlook is that of the primary disease. The ultimate failure of the circulation is often largely due to cardiac atrophy.

II. DISEASES OF THE PERICARDIUM.

i. Pericarditis.

Definition.—Inflammation of the pericardium resulting from traumatism, infection, the extension of inflammation from contiguous structures, or toxic conditions.

Etiology.—Idiopathic or spontaneous pericarditis is a purely theoretical conception. The extremely rare cases of pericarditis in children without other indications of local or constitutional disease are probably due to latent tuberculosis, or tonsillitis or other infection, or to an obscure toxæmia. Traumatic pericarditis may become the subject of medical diagnosis when the injury is from within, as in the case of the ulceration of a foreign body from the œsophagus, or injury by needles or pins that have been swallowed. Infection is the most common cause. The greater number of cases occur in connection with rheumatic fever. The pericarditis may precede the joint affection. Next in frequency are the cases due to tuberculosis. To this etiological group are to be referred the cases of pericarditis which follow blows and contusions of the chest, and those which occur in alcoholics. The pericarditis may, for a time, be the only clinical manifestation of the tuberculous infection. Less commonly pericarditis

is secondary to sepsis, especially that caused by acute necrosis or puerperal infection, or the toxæmia of scarlet fever and the other acute febrile infections.

Extension of the inflammation from the endocardium may account for the common association of endo- and pericarditis in rheumatic fever, or the later pericardial inflammation may be also a direct manifestation of rheumatism. This mode of infection is common in pleurisy and pneumonia, and may occur in œsophageal carcinoma, tuberculous or bronchiectatic cavities closely adjacent to the pericardium, tuberculous mediastinitis, perforating gastric ulcer, or subphrenic abscess. That form which sometimes occurs in purulent myocarditis, ulcerative endocarditis, aneurism of the aorta, disease of the ribs and sternum or the vertebræ arises in a majority of the cases by direct extension. Toxic pericarditis is not rarely a terminal condition in chronic nephritis, especially the interstitial variety. It is occasionally present but usually latent in gout, scurvy, diabetes, and arteriosclerosis. Among the infrequent causes of pericarditis are syphilis, carcinoma and sarcoma of the pericardium, echinococci or cysticerci, and actinomycosis. The micro-organisms most frequently encountered in the exudate are the ordinary pyogenic bacteria, the pneumococcus, and the tubercle bacillus.

The inflammatory exudate may be fibrinous, serofibrinous, hemorrhagic, or purulent. The terminal condition in cases that recover is that of more or less complete adhesion between the pericardial surfaces—*adherent pericardium*. It is customary to describe separately dry or fibrinous pericarditis, pericarditis with effusion, and adherent pericardium; but it is important to bear in mind the fact that these, in a majority of instances, are successive stages in a continuous process. Like other inflammations, pericarditis may be acute or chronic.

(a) FIBRINOUS, PLASTIC, OR DRY PERICARDITIS.

Pericarditis Sicca.

In the simple acute cases the inflammation involves first the epicardial or visceral layer; later the pericardial layer of the serous pericardium. The fibrinous exudate may be circumscribed or general. Its arrangement varies greatly. Sometimes it presents the appearance seen when two buttered surfaces are separated; sometimes there are hairy ridges in irregular parallel lines,—*cor villosum*,—or again there may be a stratified or a honeycombed appearance. There is, as a rule, a variable amount of fluid entangled in the meshes of the fibrin, but in chronic tuberculous cases with great thickening fluid is absent. The myocardium immediately subjacent is inflamed. The frequent coexistence of endocarditis is of clinical as well as etiological interest.

Symptoms.—Plastic pericarditis is sometimes latent. Even in marked cases the subjective phenomena may be indefinite. Pain is common. It is usually substernal or referred to the region of the apex. Less frequently it radiates to the neck and arm, especially on the left side. It may be stitch-like and lancinating, or dull and heavy; persistent or paroxysmal.

The fever of the primary disease may be aggravated, but the terminal pericarditis of nephritis may be unattended by a rise of temperature.

Physical Signs.—Upon inspection the signs are usually negative. The breathing may be rapid and shallow or there may be orthopnœa. Palpation in a considerable proportion of the cases reveals a more or less distinct friction fremitus. The pulse is usually accelerated—120 to 140 to the minute. The percussion borders of the heart are not enlarged in simple fibrinous pericarditis. In old cases the myocardium may undergo dilatation. AUSCULTATION.—The pericardial friction sound is most variable in character. It is frequently of a soft grazing or brushing quality. More commonly it is rubbing or grating and has been compared with the creaking of new leather. It appears to be superficial, as though produced close to the surface, and is increased by moderate pressure, in some cases obliterated by strong pressure with the stethoscope. Its intensity varies from a scarcely audible whiff to a loud coarse sound, directly appreciable to the ear. Its loudness is not dependent upon the amount of fibrinous exudate. It is sometimes absent when the fibrin is abundant, as in *cor villosum;* sometimes distinct when there is merely a thin layer. The intensity is modified by posture and undergoes remarkable changes from day to day in the course of the attack. The pericardial friction sound is usually to-and-fro, corresponding to the systole and diastole of the ventricles, but it does not bear the definite relations of endocardial murmurs to the cardiac cycle. It is sometimes single and in rare instances triple, having a somewhat irregular canter rhythm. Its systolic and diastolic portions have usually, but not invariably, the same quality and pitch. They are almost always of unequal length. It is heard over the body of the heart; sometimes most distinctly in the second, third, and fourth left intercostal spaces and adjacent parts of the sternum, sometimes at the base over the pericardial reflections upon the great vessels, and again in the region of the apex. It is usually limited to a small area, but may be distinctly heard over a large part of the pericardium. It is, however, always circumscribed and never transmitted beyond the boundaries of the heart in definite lines corresponding to the vessels, as is the case with endocardial murmurs. When pericardial effusion takes place the friction sound disappears over the body of the heart, but, except in large effusions, may still be heard in a limited region at the base.

Diagnosis.—The DIRECT DIAGNOSIS of fibrinous pericarditis depends upon the recognition of a friction sound having the foregoing characters, location, and correspondence to the revolution of the heart.

DIFFERENTIAL.—The distinction between endocardial murmurs and exocardial friction sounds is based upon the well-recognized characters of each. A double aortic murmur, particularly when accompanied by a thrill, may lead to error, but not if due heed be given to its sameness from time to time, lines of propagation, the correspondence of its systolic and diastolic elements with the cycle of the heart, and the associated arterial changes.

Pleurisy.—Pleural friction is not usually restricted to the cardiac borders of the lung, and when heard elsewhere serves to explain a pleuropericardial friction, which is due to movements of the pleural surfaces

induced by the action of the heart. This sign is by no means infrequent at the left anterior margin of the lung in croupous pneumonia, and is sometimes encountered in phthisis. It disappears upon full-held inspiration, and on ordinary breathing is more distinct during the expiratory period. Clicking and crepitant râles, occasionally heard in the region of the apex and recurring with the ventricular systole, are readily differentiated from pericardial friction.

It is stated that pericardial friction sounds are sometimes produced by milk spots on the surface of the ventricles, concretions, and in the dry condition of the tissues occurring in cholera, but such conditions are not to be confounded with true pericarditis.

Prognosis.—The course of fibrinous pericarditis as such is favorable. In some days or weeks recovery may take place without any clinical manifestation of injury to the heart. The inflammation may indeed run its course in the absence of subjective phenomena, the friction sound being the only objective sign. More commonly there are more or less urgent symptoms. The danger of serofibrinous, hemorrhagic, or purulent effusion, of implication of the myocardium with acute symptoms, and of extensive pericardial adhesions leading to chronic myocarditis invests every case with importance. As an intercurrent affection it adds to the gravity of the primary disease.

(b) PERICARDITIS WITH EFFUSION.

Pericarditis Exudativa.

There is no abrupt line of separation between dry pericarditis and pericarditis with effusion. In the meshes of an abundant fibrinous exudate there are small collections of serum; in serofibrinous effusions the pericardial surfaces are covered with fibrin, flakes of which float free in the fluid. The effusion may be serofibrinous, hemorrhagic, or purulent.

Etiologically serofibrinous effusion is usually a so-called second stage in the evolution of the attack of pericarditis, and may arise in any of the conditions in which plastic pericarditis occurs. Blood elements are present in varying amounts. Hemorrhagic effusions owe their characteristic appearance to an excess of blood. They are met with in tuberculous and cancerous pericarditis, and in those forms which occur in hemorrhagic conditions, as scorbutus and purpura, and in the aged. The quantity of blood varies from an amount only appreciable upon microscopical or chemical examination to almost pure blood. The effusion may be purulent in tuberculous cases. It is likely to be so in those due to sepsis or internal or external traumatism, or when an effusion arises in consequence of infection from a contiguous bronchiectatic cavity or vomica. The volume of the effusion is extremely variable. Experimentally the normal sac will contain without distention 150 to 200 c.c. Upon forcible distention, with compression of the heart, from 500 to 800 c.c. may be injected. The inflamed pericardium is more distensible, and with the adjustments which take place under the gradual accumulation of an effusion, as much as 1500 or even 2000 c.c. have been observed.

Symptoms.—The condition is frequently latent. There are cases in which moderate pericardial effusions run a favorable course without heart symptoms, resorption taking place in the course of two or three weeks. More commonly the early symptoms consist of chilliness, precordial pain, and fever. In well-developed cases there are two main groups of symptoms, constitutional and local. The constitutional symptoms are very often masked by those of the primary affection, and moderate terminal effusions are more frequently recognized in the post-mortem room than in the ward. In children general symptoms, as feverishness, dyspnœa, loss of appetite, fretfulness, languor, and a rapidly developing pallor, may occur in the absence of precordial pain or other symptoms suggestive of the actual condition. Pallor, weakness, insomnia, loss of appetite, dyspnœa and orthopnœa, melancholia and a disposition to suicide, and in grave cases restlessness, somnolence, delirium, and a tendency to coma are among the symptoms of pericardial effusion. The local symptoms arise from the inflammation, from the derangement of the circulation, and from pressure. The pain is referred to the precordium; less commonly to the epigastrium. It is usually sharp and lancinating; sometimes dull and aching; exceptionally it amounts only to a sense of distress and discomfort. It is usually continuous with exacerbations, but may be paroxysmal with intervals of relief. It is intensified by pressure with the stethoscope. Derangements of the circulation are manifest in cyanosis of varying intensity, shortness of breath, anxiety, and the sensation of air hunger. The patient prefers to lie upon the left side; in large effusions he is obliged to be propped up with pillows in the semirecumbent posture or to sit up in bed. The pulse is rapid, small, and frequently arrhythmic. In large effusions with thickening of the parietal pericardium the pulse may become very feeble or quite imperceptible during inspiration—*pulsus paradoxicus*. It is sometimes smaller in the left than in the right carotid and radial arteries. The circulatory symptoms are due in part to the direct pressure of the effusion upon the heart, the effects of which are greater upon the thin-walled auricles than upon the ventricles, and to the implication of the myocardium directly in relation with the inflamed epicardium. The symptoms due to pressure upon other organs are a sense of precordial oppression and weight in the epigastrium, dysphagia, aphonia, a laryngeal cough, distention of the veins of the neck, and dyspnœa from compression of the left lung.

Physical Signs.—Inspection.—In small effusions there are no distinctive signs. In moderate and large effusions the respiratory excursus upon the left side is diminished in consequence of pressure atelectasis of the lower lobe. Pericardial effusions compress the left lung to a far greater extent than the right. The epigastrium is prominent, owing to the depression of the diaphragm and liver. In children and young persons precordial prominence, widening and slight bulging of the lower intercostal spaces, and in some cases a feeble wavy cardiac impulse may be present. Palpation.—A cardiac impulse due to the contraction of the right ventricle may be feebly felt in the fourth interspace; in other cases the apex beat may be lower than normal in consequence of the depression of the diaphragm. Very often no precordial impulse can be detected. Friction

fremitus vanishes as the layers of the pericardium are separated by the effusion, except at the base, where it may sometimes be felt, especially in the erect posture. Fluctuation is not a sign of pericardial effusion. In massive effusions bulging of the left retroclavicular space has been observed and elevation of the clavicle, so that the first rib may be palpated to the sternum—*first rib sign.* PERCUSSION.—This method of physical diagnosis yields most important signs. The effusion collects first in the most dependent part of the sac and gradually rises as it increases in amount. Its presence may be first appreciated by an absence of resonance at the sternal end of the fifth right intercostal space—the cardiohepatic angle. At this point in normal and dilated hearts the vertical border of the cardiac dulness and the transverse upper border of the hepatic dulness make a well-defined right angle. In early effusion and in certain cases of obesity this angle is replaced by a curve having its concavity upward and outward toward the lung—*Rotch's sign.* As the effusion increases the precordial dulness extends

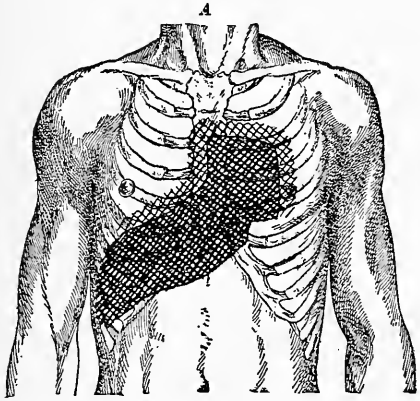


FIG. 348*A*.—Moderate pericardial effusion; quadrilateral flatness with border of relative dulness; effacement of cardiohepatic angle.

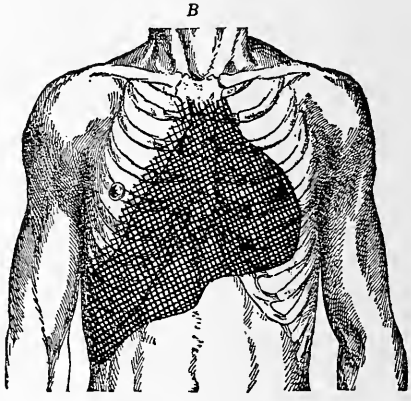


FIG. 348*B*.—Massive pericardial effusion; pyramidal area of flatness with truncated apex; right border of relative dulness; downward displacement of liver.

toward the left and upward, later toward the right, displacing the borders of the lung and forming at first a quadrilateral area of dulness with rounded corners, which with larger effusions assumes a pear-shaped outline, the larger end lying at the inferior border and extending beyond the sternal margin on the right, and beyond the position of the apex and the mid-clavicular line on the left. In large effusions the dulness may invade Traube's semilunar space. The truncated apex of this figure reaches into the upper sternal region. As the diameters of this figure are gradually reached, the area of superficial precordial dulness advances more rapidly than that of the deep or absolute dulness, until at length they nearly coincide. A circumscribed area of dulness or flatness may sometimes be found at the base of the left chest posteriorly between the inferior angle of the scapula and the vertebræ. Old pericardial adhesions, compression of the left lung, and coexistent pleural effusions greatly modify these changes in the percussion signs. AUSCULTATION.—The friction sound disappears over the body of the heart but may be heard at the base, very rarely at the apex. The first sound is obscure and indistinct; the second pulmo-

nary sound accentuated. The action of the heart is rapid and often arrhythmic. A systolic endocardial murmur may sometimes be detected. As resorption takes place the first sound becomes more distinct and the friction sound may be again heard.

Diagnosis.—The DIRECT DIAGNOSIS of pericardial effusion may be made without difficulty when the case has been seen from the outset and the above-described percussion signs have supervened upon pericardial friction sounds. Of especial value in the early recognition of effusion is Rotch's modification of the cardiohepatic angle. Very important is the triangular outline of dulness in large effusions. A sign too little appreciated is the progressive encroachment of the borders of the superficial dulness upon the area of deep dulness. The X-ray examination may be of service.

DIFFERENTIAL.—*Dilatation of the heart* very often presents extreme difficulty in the differential diagnosis. Careful clinicians have tapped the right ventricle instead of a pericardial sac distended with fluid. Dulness in the cardiohepatic angle, a quadrilateral area of dulness with rounded corners, the close approach of the borders of superficial to those of deep dulness, especially when the dulness extends to the left beyond the apex beat, constitute an association of physical signs of great importance. The truncated apex of the triangular area of dulness in large effusions, a circumscribed area of dulness near the angle of the left scapula, and signs of compression of the left lung have also diagnostic value. But most of these conditions may be present in dilated heart. In some cases of hypertrophy of the right ventricle the deep cardiac dulness due to the left ventricle extends beyond the position of the visible impulse. An undulatory impulse seen or felt in two or more interspaces, distinct though feeble heart sounds, valvular in character and having the fetal rhythm, and postural changes in the upper borders of the dulness, are signs suggestive of effusion. Overfilled veins, cyanosis, aphonia, dysphagia, and other pressure symptoms are without value in the differential diagnosis, since they may occur alike in large pericardial effusions and extreme dilatation of the heart.

Left-sided Pleural Effusion.—This condition, unless the fluid be encysted, is not often mistaken for pericardial effusion, but large pericardial effusions may closely simulate pleurisy. In the latter condition the heart is displaced toward the right, its impulse and sounds are distinct, the flatness extends around the base of the chest, the overlying compressed lung yields tympanitic percussion resonance, Traube's semilunar space is obliterated, the spleen is displaced downward and its respiratory excursus restricted, and finally dysphagia is not a pressure symptom in pleurisy. Vocal fremitus is usually distinct over an atelectatic lung, feeble or absent over fluid. The pericardial effusions which occasionally occur in pneumonia present unusual difficulties in diagnosis. The signs are masked by those of the primary lesion. There is no border-line change from dulness to clearness or tympany; at the left border of the heart the extension of dulness to the right of the sternum may be ascribed to the dilatation of a failing right heart. The modification of the cardiohepatic angle would suggest pericarditis, but it is likely to be overlooked.

The recognition of tumors of the lung, pleura, or mediastinum, or of aneurisms of the aortic or pulmonary artery, depends upon a clinical course and physical signs that are widely divergent from those of pericarditis with effusion.

The character of the fluid can be determined with certainty only by an exploratory puncture. Paracentesis of the pericardium for diagnostic purposes becomes necessary in cases progressing unfavorably. It removes an excessive accumulation of fluid and affords the basis for further treatment. When performed as a measure of treatment the gross and microscopical characters of the fluid are of diagnostic importance. Various sites are recommended for the insertion of the needle. The fourth or fifth left intercostal space near the sternum; the same interspaces to the left of the midclavicular line and within the border of the flatness on percussion; a point high in the angle formed by the ensiform cartilage and the left costal margin and the fifth right interspace 2 cm. from the sternal border when this area is flat upon percussion, are situations recommended. A small aspirator needle should be employed and strict surgical antisepsis observed. In rheumatic, renal, and tuberculous cases the fluid is usually serofibrinous; in senile, purpuric, and cancerous cases hemorrhagic; and in septic conditions it is commonly purulent. The gravity of the general symptoms usually depends upon the nature and intensity of the primary disease. When this is not the case it corresponds rather to the severity of the pericarditis, the amount of the effusion, and the rapidity with which it is formed, than to its character.

Prognosis.—The signs of effusion follow shortly the friction sound. The accumulation in rheumatic fever, nephritis, scurvy, and some septic cases is rapid, while in tuberculous cases it is usually slow. Serofibrinous effusions of moderate volume frequently undergo resorption, which may be complete in the course of four or five weeks. Reappearance of the friction sound may occur but is less common than in pleural effusions. Occasional post-mortem findings, namely, grayish material in various stages of calcareous change in the pericardium, render it probable that under certain circumstances a purulent effusion may undergo resorption. Very large effusions show little tendency to undergo resorption, and unless removed by operative measures—dissection layer by layer, or paracentesis—rapidly prove fatal by compression of the heart and other mechanical effects. Purulent effusions, unless relieved by operation and drainage, terminate, as a rule, in death. The pericarditis with effusion in scurvy, chronic nephritis, and pyæmia is almost always a terminal condition. Heart complications are frequently present. There is myocarditis involving the myocardium directly in relation with the inflamed epicardium. Endocarditis is often also present, particularly in the rheumatic cases. Old valvular lesions with associated myocardial changes may at the same time obscure the diagnosis and unfavorably affect the prognosis. When recovery takes place the pericardial surfaces become adherent. The prognosis is relatively unfavorable, as the primary constitutional condition is grave, the effusion large, and its accumulation rapid.

ii. Adherent Pericardium.

Synechia Pericardii; Obliteration of the Pericardial Sac.

Pericardial adhesions constitute a constant anatomical sequel of pericarditis, both in its latent and manifest forms. The extent of the adhesions is exceedingly variable. In many cases there are merely thread-like strings or bands of organized tissue extending from the visceral to the parietal pericardium; in others the adhesion between their membranes is universal and so close as to suggest congenital absence of the pericardium. The adjacent pleura is frequently involved and in extreme cases the heart is embedded in a dense, thick connective-tissue mass including the fibrous pericardium and the structures with which it is in relation—chronic adhesive mediastinitis.

The cases of simple pericardial adhesion may be divided into two groups:

(a) Those presenting no clinical manifestations and found upon post-mortem examination. This group includes the cases of limited adhesions and the threads and bands which scarcely affect the free movement of the heart within the sac, and some of the cases in which more general adhesions exist.

(b) Those in which, as a result of the pericarditis and adhesions, chronic myocarditis has occurred, with hypertrophy and dilatation and the symptoms of cardiac inadequacy. These cases do not always show general obliteration of the sac. A high grade of hypertrophy may occur with only partial adhesion between the layers.

Symptoms.—There are cases in which the history and physical signs are positive, but for a long period symptoms of heart disease are absent. After a time the symptoms are those of hypertrophy, then dilatation and a failing heart.

Physical Signs.—Inspection.—In young persons there may be prominence of the pericardium in consequence of hypertrophy, with an impulse visible in the fourth, fifth, and sometimes the sixth interspace and to the left of the midclavicular line. While the hypertrophy remains marked the impulse may be strong and heaving, but when it gives way to dilatation the impulse becomes more extended and undulatory, and there is systolic retraction in the neighborhood of the apex. The systolic indrawing may extend to other parts of the cardiac area. It is sometimes seen at the base of the heart and may be confined to this region. An energetic retraction in the parts about the ensiform cartilage upon the left is sometimes seen. In cases in which there are strong cardiodiaphragmatic adhesions a visible systolic retraction may be detected in the lower left ribs and interspaces behind—*Broadbent's sign.* Respiratory movement of the epigastrium may be embarrassed by the pericardial adhesions. *Friedreich's sign,* diastolic collapse of the cervical veins, is sometimes seen. Palpation.—The signs obtained by inspection are confirmed and there is often to be felt a distinct diastolic shock. The movements of the apex, under the influence of gravity, upon change of posture are less marked than under normal conditions. Pulsus paradoxus may be present. Percussion shows

an increase in the transverse diameter of the heart, and since there are usually also pleural adhesions the area of superficial cardiac dulness may not be influenced by the respiratory movements. AUSCULTATION.—The signs are not distinctive. There is very often the murmur of an associated endocarditis, especially in the rheumatic cases. With dilatation there is usually the systolic mitral murmur of relative insufficiency. Other murmurs have been described, especially a presystolic murmur, but these are inconstant and accidental.

Diagnosis.—The direct diagnosis of pericardial adhesion rests upon the history of pericarditis and the presence of the foregoing signs upon physical examination. In some cases a positive diagnosis cannot be made; in others it can be made with certainty even in the absence of a history of pericarditis. The cases that present the greatest difficulty are those in which there are adhesions between the pericardium and epicardium without adhesions to the adjacent structures; the cases in which the diagnosis may often be made with confidence are those in which there are extensive adhesions, not only between the visceral and parietal pericardium, but also between the fibrous pericardium and the surrounding parts—chronic indurative mediastinitis. Too great importance may be given to systolic retraction of the intercostal space in the region of the apex. This, in the absence of pericardial adhesions, may be due to atmospheric pressure when the energetically contracting ventricles are not followed by the border of the lung with sufficient promptness. It may also occur in hypertrophy and dilatation of the right ventricle when the left ventricle remains small. The impulse is that of the right ventricle, and to the left of it there may in some cases be seen distinct systolic retraction. As a rule, to which there are, however, exceptions, the retraction in adherent pericardium is more energetic than that induced by atmospheric pressure. When systolic retraction occurs under observation after an attack of pericarditis the diagnosis of adherent pericardium may be made. In a doubtful case the shadow cast by the Röntgen rays may be of service. It may show irregular contour of the heart, a feeble, restricted cardiac pulsation, and diminished play of the diaphragm, especially in its central parts.

Prognosis.—The outlook in simple obliteration of the pericardium depends upon the influence of the primary pericarditis and the subsequent adhesions upon the myocardium. So long as the heart retains its function the prognosis is favorable. Upon the supervention of the signs of cardiac insufficiency it becomes unfavorable. Stasis symptoms, dyspnœa, cardiac irregularity and cyanosis are of ominous import. Relief of symptoms and prolongation of life have followed surgical intervention—separation of the adherent pericardium from the sternum; removal of the sternum with its anterior periosteum, allowing the posterior periosteum to remain *in situ*.

iii. Hydropericardium; Hydrops Pericardii.

Dropsy of the pericardium occurs in connection with general dropsy in the course of renal, less frequently of heart disease, and in association with effusions into the other great serous sacs, the pleuræ and peritoneum. In rare cases of scarlet fever this condition has been observed in the absence of dropsy in other parts. Normally the pericardial sac contains a small amount of clear yellow serum, 5–10 c.c. In hydropericardium the quantity

rarely exceeds 150–200 c.c. This transudate is clear, yellowish, and may contain a few red blood-corpuscles. The pericardium is smooth and glistening. Chylous effusion is a very rare condition. Hydropericardium presents the physical signs of a moderate pericardial effusion, from which it cannot be differentiated except by the history and the associated clinical phenomena or *paracentesis pericardii*. It does not directly tend to cause death, but constitutes an additional danger in the serious affections in which it arises as a complication.

iv. Hæmopericardium.

Hemorrhage into the pericardial sac is to be differentiated from hemorrhagic pericardial effusion. It results from wounds or rupture of the heart or of an aneurism of the aorta, pulmonary artery, or coronary arteries, and has been observed as a consequence of ulceration in malignant endocarditis. As a rule, death occurs immediately with the symptoms of internal hemorrhage. In extremely rare instances the bleeding is more slow, especially in cases of minute rupture of the heart, and time permits an examination of the cases with a view to their diagnosis and treatment. The signs are then of a more or less rapidly accumulating pericardial effusion; the symptoms those of more or less abundant internal hemorrhage. The prognosis is in the highest degree unfavorable. Traumatic cases may recover after immediate operation—suturing the incised wall and draining the pericardium.

v. Pneumopericardium.

Air or gas in the pericardial sac is an exceedingly rare condition. It may result from external wounds, perforation of the œsophagus or stomach, subphrenic pyopneumothorax, a tuberculous cavity involving the pericardium, or spontaneously without solution of continuity in the sac from the presence of the Bacillus aërogenes capsulatus. The area of cardiac dulness is replaced by tympany. The impulse in the recumbent posture disappears, though it may be felt when the patient sits up. The movements of the heart are accompanied by coarse churning, gurgling noises, and the heart sounds have a loud, metallic ring, which may be heard at some distance from the chest. Fluid is usually also present.

vi. Calcification of the Pericardium.

Deposition of lime salts sometimes takes place in tuberculous pericarditis and in pyopericardium with resorption of the fluid. The condition may be partial or complete, so that the heart is encased in a sort of bony shell. Strangely enough it is usually latent, though a diagnosis of adherent pericardium has been made. The lesion is exceedingly rare.

V. DISEASES OF THE ENDOCARDIUM.

i. Endocarditis.

Definition.—Inflammation of the lining membrane of the heart.

In by far the greater number of the cases the inflammatory process is restricted to the valves—*valvular endocarditis;* exceptionally it extends to the lining membrane of the wall of the heart—*mural endocarditis.*

Two forms of endocarditis are recognized, acute and chronic.

(a) Acute Endocarditis.

Acute endocarditis is of every grade of intensity. Its milder forms run a favorable course, and the inflammatory lesions, though they impair the function of the valves, are not destructive. Its severe forms are attended with grave symptoms and usually end in death, and the lesions comprise ulceration and necrosis of the affected valves and adjacent parts. It is convenient to describe separately *acute simple or benign endocarditis*, and *acute ulcerative, infective, or malignant endocarditis*. Between these two forms there is no abrupt anatomical or clinical dividing line.

The lesions in simple endocarditis consist of minute wart-like vegetations, hence the descriptive terms *vegetative* or *verrucose endocarditis*. The left side of the heart is involved more commonly than the right, and the mitral than the aortic leaflets. These vegetations are arranged in lines upon the auricular surface of the auriculoventricular leaflets and the ventricular surface of the sigmoid cusps a little distance back of the free edges of the valves. The clinical course of the disease is determined by the subsequent changes in the valvular lesions, which may result in organization with trifling permanent alteration; in progressive sclerotic changes and deformity—*chronic valvular disease;* in the detachment of loose vegetations and embolism; or finally in an overgrowth of the vegetations and ulcerative destruction of the valve leaflets—*malignant endocarditis*. In the last, not only the leaflets but also adjacent parts may be destroyed, with perforation of a valve, the septum, or the wall of the heart, and, owing to the loosely organized character of the exuberant vegetations, multiple embolism is common.

The valve systems affected are in the order of frequency as follows: mitral alone, aortic alone, aortic and mitral together, tricuspid, and pulmonary. The walls of the heart are involved, as a rule, only in connection with the valves. Endocarditis in fetal life usually involves the right side of the heart.

Etiology. — Predisposing Influences. — Acute endocarditis, both simple and malignant, has been met with under circumstances in which no antecedent or primary disease or lesion could be demonstrated. In the majority of instances it is a secondary affection. Focal infection, tonsils, teeth, accessory sinuses, bronchopulmonary lesions, urethra, prostate, etc., should be sought for. *Simple Endocarditis.*—Rheumatic fever is by far the most common primary affection. Chorea, tonsillitis, scarlet fever, and croupous pneumonia are very frequent. It is rare in enteric fever, measles, diphtheria, variola, and varicella. In gout, diabetes, chronic nephritis, and cancer simple endocarditis is occasionally observed. Acute endocarditis is common in old cases of valvular disease—*recurrent endocarditis. Malignant Endocarditis.*—Here also rheumatic fever, pneumonia, and other acute infections play an important part as the primary disease. But it is especially in septic processes that malignant endocarditis occurs. Recurrent endocarditis is frequently malignant in type. Malignant endocarditis constitutes a grave danger in gonorrhœal infection, especially in the male. The malignant form is exceedingly rare in enteric fever, diphtheria, tuberculosis, dysentery, and scarlet fever.

Heredity plays an important rôle in the predisposition to endocarditis. There are many families in which the liability is plainly manifest in succes-

sive generations. Rheumatic endocarditis is especially common in childhood and early adult life. It may, however, occur at any age. After forty the liability to a first attack of rheumatic fever is slight. Chorea is more common in girls than in boys, and it is in accordance with this fact that the incidence of simple endocarditis is somewhat greater in females. The especial liability to sepsis which attends the child-bearing function constitutes an important predisposing influence to the graver forms of endocarditis.

Exciting Cause.—The pyogenic bacteria which are present in the lesions of the primary disease are found in the valvular vegetations and in the infected emboli common in malignant endocarditis. One or more varieties may be identified in the same case. The more common are streptococci, staphylococci, pneumococci, and gonococci. Much less frequently the bacillus of enteric fever, diphtheria, tuberculosis, and the Bacillus coli communis have been found. In the simple endocarditis of chronic diseases and cachectic states micro-organisms may be absent.

SIMPLE ENDOCARDITIS.

Symptoms.—This form very often runs a latent course without modification of the symptoms of the primary affection. In other cases increased pulse-frequency, slight irregularity in the action of the heart, a sense of precordial oppression, and attacks of dyspnœa occur. There may or may not be a rise of temperature in rheumatic cases without fresh joint affection. In young children rheumatic endocarditis may occur with trifling manifestations of illness and without arthritis, the true nature of the attack being revealed by the physical signs, the subsequent valvular disease, and recurrent attacks of well-characterized articular rheumatism. Again, to these symptoms there may be added the manifestations of acute cardiac insufficiency and grave constitutional disturbances—irregular, rapid, and feeble pulse, faintness, oppression, orthopnœa, high fever not conforming to type, profuse perspirations, and extreme pallor. Such cases lie on the border-line between simple and malignant endocarditis.

Physical Signs.—A murmur may develop at one of the valvular areas. Commonly the first sound is impure at the beginning or slightly rough. This change increases to a murmur which gradually becomes distinct. The second sound may be reduplicated, its pulmonary element accentuated. There may be slight increase in the transverse diameter of the heart and displacement of the apex to the left, signs of implication of the myocardium.

Diagnosis.—Simple endocarditis in many of the cases is discovered only by systematic routine examination. Very often it is not recognized at all. Recent endocarditis is sometimes found in cases of nephritis or carcinoma in which no murmur has been heard. When heard the murmur may be due to relative or muscular insufficiency, or to valvular disease resulting from acute endocarditis in the past. If it has developed under observation the latter possibility may be excluded but not the former. If it becomes more distinct and persists beyond the convalescence from the primary disease, a diagnosis of acute endocarditis is justified.

MALIGNANT ENDOCARDITIS.

Symptoms.—There are two groups of symptoms: those due to the primary disease or the sepsis to which it has given rise, and those due to the endocarditis. Either of these groups may dominate the clinical picture. To the first belong irregular fever, copious sweating, profound anæmia, delirium, and loss of strength; to the second group a curious air hunger, paroxysmal dyspnœa, orthopnœa, palpitation, frequent and irregular action of the heart, and the phenomena caused by emboli in various tissues and organs. A very common point of entrance for the infection is in lesions of the female reproductive organs. Injuries of the integument, boils and abscesses, suppuration of the middle ear, inflamed hemorrhoids, gonorrhœa in the male, croupous pneumonia, and suppurative disease of the liver or of bone are frequent causes. Old valvular disease of the heart is very common. The anamnesis is of great value in the diagnosis. In a doubtful case this ground must be carefully gone over. Septic, typhoid, cerebral, and cardiac forms are described, but the picture is a very diverse one and the distinctions are by no means clear. The Septic Form.—There is usually a history of puerperal infection, a neglected wound, acute necrosis, or gonorrhœa. Severe rigors, irregular pyrexia, colliquative sweating, and vomiting are common. Heart symptoms are sometimes subordinate and the signs overlooked. Embolism is common. The Typhoid Form.—The temperature is high and subcontinuous or remittent in type. There are great depression, diarrhœa, sometimes tympany, drenching sweats, delirium, somnolence, and a tendency to coma. Heart symptoms are often obscure. Murmurs may be absent. The Cerebral Form.—The onset is abrupt with the signs of a basilar or cerebrospinal meningitis. Sudden violent delirium is followed by coma. The Cardiac or Recurrent Form.—This variety occurs in individuals who are the subjects of chronic valvular disease. The symptoms are very variable. The attack may run a rapidly fatal course with septic or so-called typhoid phenomena and high fever, or recovery take place after several weeks. Repeated attacks with the clinical manifestations of an acute endocarditis may occur.

Endocarditis Lenta.—Under this term a form of malignant endocarditis due to *Streptococcus viridans* has been described. There is more or less continued fever, with chills, sweating and irregular remissions and exacerbations. Pallor, sallowness, petechiæ, tender cutaneous nodules upon the hands and feet are present. Albuminuria with occasional urinary blood signs is common. Leucocytosis is constant. The physical signs of an acute endocarditis are usually but not invariably present. Blood cultures show the presence of *S. viridans*. Signs of septic infarction of various viscera occur. The progress of the disease is slow and the termination in recovery extremely rare. Post-mortem findings comprise in addition to the lesions of malignant endocarditis, those of infarction of the kidneys, spleen, brain and other organs.

Embolism may cause the most diverse manifestations, among which are delirium, coma, hemiplegia, monoplegia, and central derangements of vision and hearing in consequence of implication of arterial branches in the brain or meninges; pain in the splenic area from infarction and perisplenitis; pain in the lumbar region and bloody urine from infarction of one or both the kidneys; and abscesses in the subcutaneous tissues, which are often

multiple. The last are common in the legs and feet, less so in the arms, occasional in the buttocks or shoulders, and infrequent in the face or neck. To this cause must also be ascribed the retinal hemorrhage which sometimes occurs, and the rare complication of suppurative panophthalmitis. Erythematous and petechial rashes are common. Jaundice occasionally occurs. As in other forms of sepsis, diarrhœa is often troublesome. Leucocytosis is usually present.

Physical Signs.—There are no signs of importance upon inspection. Palpation yields valuable information as to the character and extent of the impulse. Thrills in the mitral and aortic areas may be felt. Percussion shows the heart to be moderately enlarged in its transverse diameter, especially to the right of the sternal border—dilatation of the right ventricle. Upon auscultation the signs are by no means constant. Errors in diagnosis may be avoided by bearing in mind the fact that in a considerable proportion of the cases no murmur can be detected upon careful search. Usually, however, there are well-marked, often harsh, murmurs in the mitral and aortic areas, mostly systolic, but frequently also presystolic or diastolic as the case may be, and often changing their quality, rhythm, and intensity from time to time. This variability in the murmurs and in thrills when present corresponds to changes in the dimensions and other physical characters of the lesions, and constitutes a diagnostic sign of the highest importance.

Diagnosis of Malignant Endocarditis.—Direct.—In the absence of the physical signs of endocarditis and of embolism the recognition of the disease may be impossible. Sepsis, associated with murmurs which vary in character and intensity, or the signs of embolism form the basis for a positive diagnosis.

Differential.—The following conditions are to be considered: *Acute Simple Endocarditis.*—The general symptoms in the malignant form are much more intense. Recurrent chills, irregular pyrexia, and profuse sweating occur. Embolic processes are far more common. There are borderline cases which may be referred to either category, but in these the absence of a focus of infection is in favor of a severe form of the benign type of the disease. In the malignant cases leucocytosis, petechial eruptions, and the urinary findings of acute nephritis are of diagnostic importance. Blood cultures may yield conclusive results. *Enteric Fever.*—Many of the cases are at first regarded as irregular forms of enteric fever. The gradual rise of temperature in the latter disease, the slowness of the pulse in proportion to the pyrexia, the greater enlargement of the spleen, the rose rash, and a positive Widal reaction constitute a symptom-complex not seen in any other affection. It is true that a rose spot or two may sometimes be found in a septic case. Grave cases of enteric fever with secondary infection may become distinctly septic and develop malignant endocarditis. *Typhus Fever.*—This now infrequent disease usually occurs in local outbreaks, and is characterized by early intense headache, stupor, a peculiar petechial rash appearing about the fourth day and all over the body except the face, and an average course of about fourteen days. *Hemorrhagic Smallpox.*—This rare malignant variety of variola occurs only in the unvaccinated and has little in common with ulcerative endocarditis except its profoundly infectious nature and rapidly fatal issue. *Malarial Fever.*—In some of the cases of

malignant endocarditis the ague-like paroxysms of chill, fever, and sweating recur with a periodicity suggestive of malarial infection. The absence of the blood parasite and the total failure of quinine to influence the progress of the disease are conclusive.

Prognosis.—The immediate outlook in the simple form of acute endocarditis is favorable. In the majority of instances, however, it proves to be the point of departure for chronic valvular disease. The remote consequences are therefore often grave. An attack in early life may prove the cause of protracted and irremediable disability and ill health. The prognosis in the malignant form is highly unfavorable. Most of the cases end in death. Those that recover are of the cardiac type and recurrences are common. The duration of malignant endocarditis varies from a few days to several weeks.

(b) Chronic Endocarditis.

Definition.—Connective-tissue new formation in the valvular endocardium, having its beginning in, (a) acute endocarditis, (b) the extension of arteriosclerosis from the arterial system, or (c) occurring as a primary affection and leading to various deformities of the valves and impairment of their function.

(a) The vegetations and thrombi become organized, with the production of nodular fibroid thickening at the margins and later throughout the substance of the leaflets. The connective-tissue overgrowth undergoes contraction, with thickening, incurving of the edges of the leaflets, and other coarse deformities. This process affects the left side of the heart and the mitral valve system more frequently than the aortic. (b) The sclerotic change in the valves arises independently of an antecedent acute endocarditis, and is one of the manifestations of a general or more or less extensive fibroid transformation affecting the arterial system—arteriosclerosis. In antenatal life the right heart is usually affected; after birth the left heart. This process generally involves the aortic valves but often extends to the mitral, and in rare instances affects the mitral without implication of the aortic. (c) Primary sclerosis occurs as the result of habitual prolonged and severe muscular effort. The aortic valves especially suffer in this form of chronic endocarditis. The toxins of syphilis and gout, the intoxications of lead and alcohol, prolonged anxiety, grief and worry, and the tissue changes incident to old age are credited with the production of sclerotic changes in the valves. Whatever the mode of origin the result is the same, deformity and impairment of function.

The deformities are various and arise from thickening, curling, adhesions, superficial necrotic changes, the deposition of lime salts, loss of elasticity, and the stretching of parts still capable of yielding to pressure. The papillary muscles show sclerotic changes, particularly at their tips. The chordæ tendineæ are shortened and thickened and in some instances destroyed. Chronic mural endocarditis may be due to myocardial changes.

The effect of the valvular lesions is insufficiency or stenosis, which may be single or combined.

The derangement of function in both insufficiency and stenosis consists in an interference of the normal course of a part of the blood stream. In insufficiency the affected blood is permitted to flow back through the

orifice—regurgitation; in stenosis it is held back at the orifice. In the combined lesions some of the blood is held back and some passes back. The altered valves can neither be completely closed nor fully opened. The overfilled chamber and increased resistance demand increased work on the part of the heart, and this leads to hypertrophy. When the increase in work and the increase in power are equal the balance of the circulation is maintained and the lesion is said to be compensated. The tendency on the part of the valvular lesion is to progress. That on the part of the compensating hypertrophy is to advance at an equal rate. Thus compensation advances hand in hand with the lesion, and symptoms are absent. This process goes on, however, at the cost of corresponding impairment of the reserve power of the heart. The capacity for extraordinary work is progressively impaired. A sudden violent effort, hill climbing, worry, the stress of life, an acute illness reveal beginning cardiac inadequacy. It is fortunate that in the physical signs of valvular disease we have, while compensation is still maintained, the means of recognizing the condition and can institute measures to avert disaster. There are cases, however, in which compensation does not occur. The lesion is too great or has developed too rapidly, or the myocardium is unsound, and dilatation takes place at once.

After a time the compensation becomes impaired. This change may be due to further advance in the valvular lesion, with which the heart muscle is unable to keep pace, or to the insufficiency to which the hypertrophied muscular tissue is peculiarly prone. The manifestations are not different from those of chronic myocarditis due to other causes. They vary progressively in degree. Hence the terms impaired compensation, broken or lost compensation, de-compensation.

It is in valvular disease that a functional diagnosis is of the highest importance. Not so much what is the lesion or the valve system involved, as how it affects the function of the heart, is the question in the individual case. The condition of the heart muscle is far and away more important than the valvular lesion. Is the compensation maintained or impaired? This is the main point. If impaired, to what extent? Upon the reply to these questions the management of the case and the future of the patient depend.

When the valvular lesion is compensated the arterial pressure is normal. Under ordinary circumstances there is no dyspnœa upon moderate exertion. Cyanosis is not present. The liver is not enlarged, and the normal amount of urine is voided. We think too much of the condition of the valves; too little of that of the myocardium. The former is beyond the reach of prophylaxis and cure. Intelligent attention to the latter means, in many cases, the relief of distressing symptoms and the postponement of disaster. When compensation fails the heart is enlarged toward the right, there is dyspnœa upon slight exertion or even at rest, orthopnœa, faint cyanosis, enlargement of the area of liver dulness, a feeble impulse, and a small, rapid, often irregular pulse,—all manifestations of cardiac inadequacy.

About 75 per cent. of the cases are due to acute endocarditis, about 12 per cent. to arteriosclerosis, and the remainder to primary valvular selerosis and other causes. Of the cases resulting from acute endocarditis nearly 60 per cent. are due to rheumatic fever. The distribution of the lesions in valvular disease following rheumatism is, according to Romberg,

as follows: mitral about 59 per cent.; mitral and aortic 29 per cent.; aortic alone, 9 per cent.; and mitral and tricuspid, and with these the aortic and pulmonary, 3 per cent. As to age, most of the cases originating in rheumatic endocarditis are first recognized between the tenth and thirtieth years. The chronic endocarditis of early life is mostly due to acute endocarditis; that of advancing years to sclerosis. The two sexes are liable nearly to the same degree. Other predisposing influences are unimportant.

VI. CHRONIC VALVULAR DISEASE.

i. Aortic Insufficiency

Aortic Incompetence; Aortic Regurgitation; Carrigan's Disease.

The valves fail to close the aortic orifice and a portion of the blood that has passed into the aorta with the systole returns to the ventricle during diastole.

The loss of function is, in a great majority of the cases, the result of deformity of the valves; in others it is due to dilatation of the aortic ring—*relative aortic incompetency.*

The deformity may be, (a) congenital, and arise from the fusion of two semilunar leaflets at their lateral borders, or from a narrow slit parallel with and close to the free edge. Such valves frequently show sclerotic changes. (b) The result of acute endocarditis in which the insufficiency is caused by the vegetations, or by ulceration and necrosis, or by adhesions with the later changes which attend sclerosis. (c) The manifestation of progressive sclerotic processes, thickening, rigidity, incurving at the borders, and shortening of the valves. (d) Rupture of a valve segment, an accident due to excessive muscular strain, probably never occurring in previously sound valves and very infrequent in disease, if the ulcerated and necrotic valves of malignant endocarditis be excepted.

Dilatation of the outlet may occur in arteriosclerosis involving the aorta immediately above the outlet, in aneurism of the ascending portion of the aortic arch, and in advanced age as a senile change. In aortic insufficiency due to acute endocarditis there is frequently also some degree of stenosis; in the form associated with arteriosclerosis narrowing is comparatively rare.

Etiology.—Aortic insufficiency may occur at any age. It is, however, chiefly met with in middle life and is far more common in males than in females. Rheumatic fever and other acute infections associated with acute endocarditis, conditions which favor arteriosclerosis, as occupations involving continuous and prolonged excessive muscular effort, injudicious devotion to athletics, poisons such as lead and alcohol, and gout and syphilis are important etiological factors.

Direct Effects upon the Heart and Vessels.—The reflux of blood causes overdistention of the left ventricle and diminution of the normal amount in the aorta and its branches. The failure of the valves to close deprives the blood in the arterial tree of its normal base of support, which is transferred in a degree corresponding to the valvular defect to the ventricular wall. The cavity of the ventricle is overdistended. Dilatation occurs and is followed by hypertrophy. In the sclerotic forms the compensation

follows the lesion and symptoms do not for a time occur. In the suddenly developing cases—ulcerative lesions, rupture—compensation does not occur, and the gravest symptoms of acute dilatation of the heart immediately follow. The cardiac hypertrophy and dilatation are often extreme. There may be associated lesions of the mitral leaflets. Relative mitral insufficiency results from the enlargement of the mitral ring. The left auricle thereupon undergoes dilatation and hypertrophy, and as the case progresses similar changes take place in the right chambers of the heart. With each systole the dilated and hypertrophied ventricle sends into the arteries an increased amount of blood with augmented force. There is immediate but momentary widening and elongation of these vessels visible in their superficial branches—*locomotor pulsation.*

Symptoms.—Compensation may be fully maintained for a long time. Pain is among the earlier symptoms. It is sometimes dull and limited to the precordia; sometimes sharp and paroxysmal, radiating to the neck and left arm. Angina pectoris is common. Anæmia is also a comparatively early manifestation.

As compensation fails, symptoms of cerebral anæmia occur upon sudden effort, rising from bed, or in the act of defecation. Among these are headache, vertigo, phosphenes, and faintness. Presently to these are added precordial distress, and sometimes palpitation, dyspnœa, and œdema of the feet. Cyanosis is not common and blood-streaked sputa less frequent than in other forms of chronic valvular disease. Insomnia and annoying dreams, delirium and hallucinations, and a suicidal tendency are symptoms of the later stages. Irregular fever and embolism in various arterial distributions may be the manifestations of an intercurrent acute endocarditis.

Physical Signs.—Inspection yields characteristic signs. This is a valvular disease which may often be recognized, when the patient is stripped to the waist, by inspection alone. There are the evidences of a high grade of cardiac hypertrophy, namely, dislocation of the apex beat to the left and downward occasionally as far as the line of the anterior axillary fold and the seventh or eighth interspace; a widely extended heaving impulse; prominence in the precordial area, especially in young persons; throbbing at the root of the neck. The superficial arteries abruptly expand and almost as suddenly collapse. With each pulsation they are thrown into sinuous curves, which are conspicuous in the temporals, brachials, and radials. The pulsating aorta may be seen in the episternal notch and in the epigastrium. Capillary pulsation follows the line drawn upon the forehead with the finger-tip, or may be seen in the finger-nails, or, in marked cases, it may occur at times spontaneously in the hands and face. Venous pulsation is sometimes visible, especially in the large veins of the back of the hands. The pulse has the peculiarities of the Corrigan or water-hammer pulse. It is appreciably retarded, large, quick, and rapidly receding. It is for this reason spoken of as the *collapsing pulse.* This last peculiarity is intensified when the hand is raised. Upon palpation a forcible impulse may be located in two or three intercostal spaces, and general heaving of the chest perceived by palpation with the whole hand. A

diastolic thrill may sometimes be felt. Systolic depression, when present, is not so often the sign of pericardial adhesion as of atmospheric pressure. Upon ophthalmoscopic examination the retinal arteries are seen to pulsate. In aortic regurgitation of high grade there is sometimes seen distinct backward nodding of the head corresponding to the systole. Upon percussion, since the hypertrophied heart pushes the lung before it, the area of superficial dulness and that of deep dulness are alike greatly increased, especially downward and to the left. In extreme enlargement of the left ventricle the right heart is displaced to the right.

Upon auscultation there is to be heard at the base of the heart and downward a diastolic murmur, caused by the reflux of blood through the insufficiently guarded aortic orifice into the ventricle. This murmur is often faint at the aortic cartilage, but usually distinct or loud at the sternal end of the third left intercostal space or third cartilage, over the seat of the valve, and propagated along the left border of the sternum to the ensiform cartilage. The diastolic murmur of aortic insufficiency is, in many cases, more distinct at the pulmonary

A

Fig. 349*A*.—Aortic regurgitation; carotid tracing.

B

Fig. 349*B*.—Aortic regurgitation; radial tracing.

than at the aortic *punctum maximum*. It usually is loud at the beginning and rapidly becomes fainter and lasts throughout the period of diastole. The murmur of aortic insufficiency may, in some cases, be more distinct in the recumbent than in the erect posture. The second aortic sound is either wholly inaudible, being replaced by the murmur, or faintly heard over the aortic cartilage, or finally, when absent at that point, it may be heard over the carotid. The first sound may be normal at the base. A systolic murmur is not always the sign of combined stenosis. It is commonly short, and when coarse and accompanied by a thrill it may be the sign of rigid lesions projecting into the aortic space without actual narrowing, or of abrupt aortic dilatation. While compensation remains good, the first sound at the apex is normal or simply intensified and prolonged. When it fails, the systolic murmur of relative mitral insufficiency is heard.

Flint's Murmur.—A coarse, rumbling murmur, presystolic in time, heard in a limited area just above the apex, and accompanied by a thrill. This murmur has the same qualities and time relation to the cardiac revolution as the murmur of mitral stenosis, but it is not associated with the sharp first sound, the abrupt impulse, and the pulsation in the second and third

interspaces which characterize well-marked cases of mitral stenosis. It is not heard continuously, but comes and goes under conditions not well understood, and is met with in a large proportion of the cases of uncomplicated aortic insufficiency.

Over larger arteries, and especially over the femoral, there is sometimes heard a double murmur—*Durozicz's murmur*. The arteries between the pulse-beats are abnormally empty and soft. The systolic blood-pressure is high and the diastolic pressure abnormally low. The sphygmogram is characterized by abrupt high ascent, sharp summit, and faintly marked dicrotic notch.

Diagnosis.—The DIRECT DIAGNOSIS of aortic insufficiency rests upon the presence of a diastolic murmur, cardiac hypertrophy, and the pulse of Corrigan—*pulsus celer*. It finds support in the occurrence of Flint's murmur, or an associated aortic systolic or mitral systolic murmur—relative insufficiency—and in the tendency to massive hypertrophy. There are cases in which the diastolic murmur cannot be heard, or a systolic murmur only is present, yet the pulse and cardiac hypertrophy point to insufficiency of the aortic valve. In high fever with great loss of arterial tone, intense anæmia, some cases of hysteria and neurasthenia, and the more acute forms of exophthalmic goitre, the conditions of the peripheral circulation are very suggestive of aortic insufficiency, but in all these the history of the case and the concomitant symptoms and physical signs are of help in the differential diagnosis.

Prognosis.—The compensation may be maintained for years without symptoms referable to the heart, and the patient lead a fairly active life. The outlook is better when the valvular defect follows acute endocarditis and develops early in life. Associated mitral lesions are unfavorable. Relative mitral insufficiency, by which the arterial conditions are modified, tends to transfer the stress of compensation from the left ventricle to the auricle and thence to the right heart. Sudden death is a danger. It often occurs without marked previous symptoms of heart disease. When compensation fails more gradually, the characteristic symptoms of progressive cardiac inadequacy arise.

A very large proportion of the cases of aortic insufficiency are due to syphilitic aortitis. The Wassermann test is therefore a necessary diagnostic procedure. If the reaction be positive the likelihood of a coexisting aneurism is to be considered.

ii. Aortic Stenosis.

Aortic Obstruction.

The aortic outlet is narrowed or constricted. A portion of the blood which should pass into the aorta with the ventricular systole is held back with every revolution of the heart. That function of the valve which consists in the retreat of its segments into contact with the wall of the aorta before the blood stream, is impaired. This affection is comparatively rare.

The lesion may be, (a) congenital, in which case the cusps may be united to form a thin diaphragm-like membrane with a small slit-like opening, or there may be a subvalvular stenosis the result of prenatal endocarditis; (b) the result of an acute endocarditis with adhesions, stiffening, and vegetations which have undergone fibroid and calcareous changes; or (c) the outcome of an arteriosclerotic process. Very often the

last are associated with extensive atheromatous changes in the aorta, and the sigmoid valves are buttressed out by rigid calcareous masses in the sinuses of Valsalva. Under these circumstances the blood supply to the coronary arteries is diminished and myocardial degeneration hastened.

The obstruction to the outflow of the blood throws increased work upon the left ventricle, which undergoes hypertrophy. Thus compensation is established. So long as compensation is maintained, the ventricle undergoes little or no dilatation, but it appears small in view of the great thickness of the wall and is, therefore, sometimes spoken of as concentric hypertrophy in contradistinction to the ordinary eccentric hypertrophy seen in aortic or mitral insufficiency. When compensation fails the left auricle undergoes dilatation and hypertrophy of its wall, there is increase of the blood-pressure in the pulmonary circuit and stress upon the right ventricle.

Of aortic stenosis it is especially true that the valves can neither fully open nor completely close. Combined stenosis and insufficiency are therefore common; uncomplicated stenosis is rare.

Relative stenosis is that condition in which with a normal aortic ring and valve cusps there is abrupt dilatation of the aorta immediately beyond.

Etiology.—This is a rare valvular affection. Its incidence is greater in males than females, and it occurs with more frequency in old men with atheromatous arteries than at an earlier period of life.

Symptoms.—With fair compensation there are no special symptoms, and aortic stenosis may reach a high grade without marked evidences of derangement of the general health. Among the symptoms which attract the attention of the patient to his circulation are those indicative of transient cerebral anæmia—vertigo and faintness. In some cases epileptiform seizures have been observed. Palpitation and precordial pain are less common. As compensation fails the symptoms of cardiac inadequacy are progressively developed.

Physical Signs.—Inspection shows, as a rule, a heaving impulse due to the left ventricle hypertrophy, situated at the normal place or slightly to the left. As compensation fails and dilatation of the left and later of the right ventricle takes place, the impulse is displaced beyond the mid-clavicular line. A distinct thrill corresponding in time and duration to the systolic murmur may be detected at the sternal border or at the right side of the root of the neck over the carotid. The pulse is somewhat retarded. In other respects, namely, as to volume and tension, it often preserves its normal characters, though in stenosis of high grade it may be small and slow, with the filling of the arteries well maintained between the beats. The sphygmogram shows a slow rise, a broad summit, and a slow decline. There is in most of the cases in advanced life evidence of marked arteriosclerosis. Upon palpation the position of the apex beat may be obscured by pericardial adhesions or an

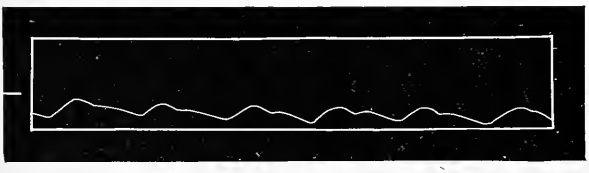


Fig. 350.—Aortic stenosis; radial tracing.

emphysematous lung. Upon percussion while compensation is still maintained, the transverse diameter of the absolute dulness and that of the relative dulness of the heart are little if at all increased. Auscultation discloses in the second right intercostal space at the sternal border a very distinct systolic murmur, usually coarse and harsh. This murmur is among the loudest of the heart murmurs and may sometimes be heard at a distance of some feet from the patient. Not infrequently it has a musical quality during some part of its course. It is distinctly transmitted to the carotids and subclavians, especially upon the right side; less plainly over the heart, but in some cases it may be heard at the apex. Very characteristic is the absence of the second aortic sound. A second sound heard at the aortic cartilage is in most cases transmitted from the pulmonary valve. When compensation fails the murmur may be faint and distant and the thrill disappear.

Diagnosis. — DIRECT. — Aortic stenosis may be recognized by the association of a loud, rough, or musical systolic murmur having its point of maximum intensity at the aortic punctum maximum, and accompanied by a thrill, the signs of hypertrophy of the left ventricle, an inaudible or faint aortic second sound, and a slow, regular pulse of moderate tension.

DIFFERENTIAL.—Errors of diagnosis are common. They arise from attaching too great importance to a systolic murmur in the aortic area in the absence of actual signs of lesions of the valve and hypertrophy of the ventricle. The following conditions in which such a murmur may be heard are to be considered: *Sclerosis of the aorta* directly beyond the valve, or of a cusp without narrowing of the orifice may, particularly when associated with the cardiac hypertrophy of nephritis, closely simulate aortic stenosis. In favor of the former condition would be an accentuated aortic second sound, and a small, regular, and rather slow pulse. *Aneurism of the Ascending Portion of the Arch.*—A history of syphilis or strain, pressure symptoms,—as pain, dyspnœa, or cough,—inequality of the pulses, displacement of the heart as a whole rather than hypertrophy of the left ventricle, circumscribed dulness and bulging with or without a thrill, tracheal tugging, and diastolic shock, all or several, when present, justify a diagnosis of aneurism. An X-ray examination may be of great service in a doubtful case. In *anæmic conditions* the basic murmur is often loudest in the aortic area. This soft bruit is very different from the loud, harsh murmur of stenosis; the aortic second sound is heard, there is not usually hypertrophy of the left ventricle, the pulse is more frequent, and anæmia may be demonstrated upon examination of the blood. *Relative aortic stenosis* may be suspected when, with a systolic aortic murmur, the second aortic sound is preserved, left ventricle hypertrophy is lacking, and signs of dilatation of the aorta are found upon percussion or by palpation with the finger-tip in the episternal notch.

Prognosis.—Cases following acute endocarditis in early life with good compensation may go on without cardiac symptoms for many years. Those due to arteriosclerosis beginning in advancing life are of much less favorable outlook. The changes are essentially progressive, the coronary arteries are liable to become involved, and with the development of myocarditis compensation fails. This form of chronic valvular disease is not attended with an especial liability to sudden death.

iii. Mitral Insufficiency.

Mitral Incompetence; Mitral Regurgitation.

A portion of the blood in the left ventricle, which upon systole should pass onward into the aorta, leaks back through the auriculoventricular orifice into the left auricle. Mitral insufficiency is the result of valvular disease, or it may occur without lesions of the valves in consequence of dilatation of the heart—*relative insufficiency*—or of derangement of the mechanism by which the leaflets are brought into effectual coaptation—*muscular insufficiency.*

Mitral Insufficiency Due to Chronic Valvular Disease.—The structural defects in the valves are in the great majority of the cases the result of acute endocarditis; infrequently the outcome of primary sclerotic processes. They consist of an overgrowth of fibroid tissue with thickening and shortening of the segments, adhesions between their borders, shortening of the chordæ tendineæ, and the deposition of lime salts in the new-formed tissues. In advanced cases the altered valves are often transformed into a thick, rigid calcareous diaphragm perforated by an irregular oval opening. Owing to the nature of the lesions uncomplicated mitral insufficiency is rare, the condition being, as a rule, associated with some degree of stenosis.

The effect upon the heart is as follows:

(a) With each ventricular systole a quantity of blood, varying according to the valvular defect, is returned from the ventricle to the left auricle. This results in an overdistention of the auricle with dilatation and hypertrophy.

(b) The left ventricle undergoes dilatation in consequence of the increased volume of blood received from the overfilled auricle. It, however, empties itself in the normal time and becomes hypertrophied to meet the increased work. Notwithstanding the amount of blood returned to the auricle, the normal quantity enters the general circulation.

(c) In the minor degrees of mitral insufficiency, the dilatation and hypertrophy of the left chambers of the heart suffice for compensation.

(d) In higher grades, the increased blood-pressure due to the overfilling of the left auricle is transferred to the pulmonary veins, and by way of the capillaries to the branches of the pulmonary artery, and thence to the right ventricle, which in turn undergoes hypertrophy.

(e) So long as the compensation thus established is maintained the right ventricle does not undergo dilatation.

(f) The prolonged overfilling of the pulmonary vessels produces brown induration of the lungs.

(g) The compensation may be indefinitely prolonged, but after a time it fails, the left ventricle no longer discharges the normal amount of blood into the aorta, the right ventricle begins to dilate, there is relative insufficiency of the tricuspid valves, and the right auricle becomes dilated. The pressure is transferred to the venous system, and the passive visceral congestions, dropsies, anæmia and other symptoms of cardiac dyscrasia begin to show themselves.

Etiology.—Mitral insufficiency is the most common of the chronic valvular diseases. Predisposing Influences.—These are found chiefly in the acute infections, in the course of which acute endocarditis occurs, especially rheumatic fever. Age, therefore, also constitutes an important predisposing factor, mitral valvular disease being especially a disease of childhood and early adult life. It may be said that the valvular diseases of early life are of inflammatory origin; those of advanced life sclerotic. Sex appears to be wholly without influence.

Exciting Cause.—The immediate cause of the deformity of the valves is to be found in the advance of lesions having their point of departure in previous inflammation or sclerosis.

Symptoms.—While compensation is maintained there are no distinctive symptoms. With insufficiency of high grade there may be merely slight dyspnœa upon unusual effort, and a rather marked tendency to attacks of bronchial catarrh. When compensation is incomplete the symptoms are very suggestive. Faint cyanosis, dilated superficial venules, dyspnœa and palpitation upon moderate effort, and frequently recurring bronchitis, often accompanied by blood-streaked sputa or hæmoptysis, constitute the clinical picture. Nevertheless, such patients often continue to work and take pleasure in life for a long period.

With broken compensation the disability is complete. The symptoms are those of advanced cardiac inadequacy, palpitation, feeble, irregular heart action, an irregular, feeble pulse, arrhythmia, dyspnœa, and harassing cough with thin blood-stained sputa containing alveolar cells with pigment granules—*Herzfehlerzellen*. Precordial distress and a sensation of heart failure are common. Another group of symptoms comprise those due to passive congestions. Among these are pallor, faint cyanosis, yellowness of the skin, drowsiness, insomnia, dropsy beginning at the ankles and rising to the body with accumulations in the loose tissues around the pudenda, in parts that are dependent, as the buttocks and flanks, and in the serous sacs, and diminished urine with albumin, casts, and blood-corpuscles. Even at this stage compensation may be by rest and treatment to some extent restored, only to be lost again in a little while. Death is not usually sudden, although at the last it may come quickly. Recurrent endocarditis is common and frequently of the malignant type. Subacute pericarditis is common. Among the intercurrent diseases to which the patients appear to be especially liable are bronchitis, bronchopneumonia, pleurisy, pulmonary infarct, and cerebral embolism. There is also a marked tendency to cerebral and retinal hemorrhage and to epistaxis. Febrile attacks are common in the absence of assignable cause. But the rise in temperature may often be explained by demonstrable infectious or septic conditions.

Physical Signs.—With fair compensation the cardiac enlargement is moderate. The impulse is displaced to the left and stronger than normal. When the compensation is broken it is extended, undulatory, and feeble. Palpation determines the force and extent of the impulse. With compensation it is forcible and heaving; in failure it is feeble and extended. In a small proportion of the cases a faint systolic thrill may be detected at the apex. The pulse with compensation is full and regular, but it may be of low tension. As compensation fails it becomes small, feeble usually,

somewhat increased in frequency, and arrhythmic. The irregularity persists when compensation is restored, and the pulse of mitral insufficiency once irregular is almost always irregular. The transverse dulness is increased toward the left and to some extent upward. It does not usually extend to the right while compensation is maintained. Exceptionally in large hypertrophy of the left ventricle the right ventricle may be displaced beyond its normal position toward the right. With failing compensation the dilated right ventricle gives increased dulness to the right of the sternal border. Upon auscultation there is heard a systolic murmur having its point of maximum intensity at the apex and being transmitted in all directions, but most distinctly in the direction of the axilla. In

A

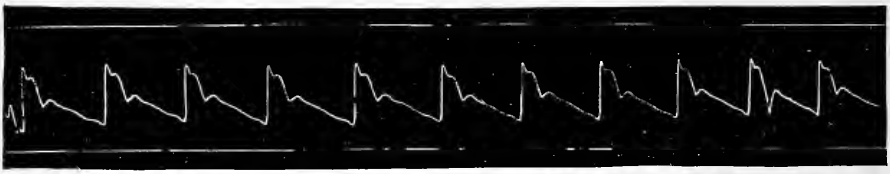


Fig. 351*A*.—Mitral regurgitation; good compensation; radial tracing.

some cases this murmur is loudest along the left border of the sternum or in the pulmonary area. It may also be heard along the lower part of the inner border of the left scapula. It is sharp, less frequently soft and blowing, and sometimes musical, especially toward the end. Its intensity varies from time to time. It may be more distinct in the erect than in the recumbent posture, but it is usually audible in both

B

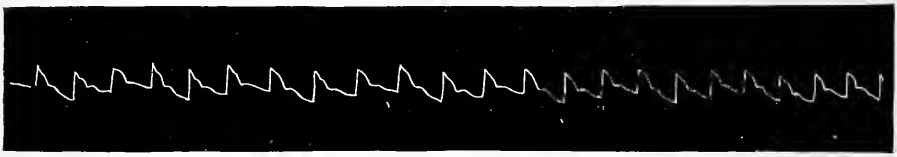


Fig. 351*B*.—Mitral regurgitation; radial tracing.

attitudes. It may accompany or wholly replace the first sound of the heart. The second pulmonary sound is accentuated. A more or less distinct presystolic murmur is the sign of a combined stenosis but it is by no means always to be heard.

Relative and Muscular Insufficiency. — (a) Relative insufficiency of the left auriculoventricular valve—*mitral valve system*—results from overdistention of the auriculoventricular ring. It occurs in acute dilatation of the heart,—*heart-strain;* the heart starvation of acute illness and chlorosis or anæmia,—and is then described as primary; and in aortic regurgitation and aortic stenosis upon failure of compensation, when it is known as secondary relative insufficiency.

The etiological factors are those of acute or gradual dilatation. The pathological condition is stretching of the ventricular ring; the derangement of function, incompetence of the valve system. The rational symptoms and physical signs are those of valvular insufficiency.

(b) Muscular insufficiency may involve the mitral or the tricuspid valve system. It is due to a derangement of the mechanism by which the closure takes place. There may be evidences of moderate dilatation of the auriculoventricular ring, but these are often absent. Pathological changes in the myocardium are, however, present. There is myocarditis involving the ring muscle at the base of the ventricle and the papillary muscles. The occasional occurrence of transitory systolic mitral murmurs in otherwise healthy men, in the absence of a history of acute cardiac dilatation, justifies the assumption that muscular insufficiency may sometimes be purely functional. It may aid the understanding of this somewhat obscure subject to recall the fact that the auriculoventricular valves are held in close contact during systole, not merely at their margins but throughout their auricular faces, by the pressure of the blood upon their ventricular surfaces.

Diagnosis.—The DIRECT DIAGNOSIS of mitral insufficiency may be attended with difficulty. A systolic murmur having its point of maximum intensity at the apex, propagated to the axilla, and heard at the angle of the scapula; accentuation of the pulmonary second sound; and the signs of hypertrophy of the left ventricle, namely, increase in the transverse diameter to the left, and a strong impulse, are important but not in every case conclusive.

The DIFFERENTIAL DIAGNOSIS between valvular and relative and muscular insufficiency cannot always be made. Here the anamnesis is very useful. In the latter groups of cases a history of acute illness, as diphtheria, enteric fever, or influenza, but no history of recent rheumatism or scarlet fever, recent blood loss or other cause of secondary anæmia, and primary anæmias, as chlorosis or pernicious anæmia, are very suggestive. The signs of arteriosclerosis and of chronic nephritis must be considered when the diagnosis is obscure. These conditions are frequently associated with myocarditis and muscular insufficiency. The very frequent association of some degree of stenosis with insufficiency gives to a presystolic murmur and thrill great value in a doubtful case.

Prognosis.—Mitral insufficiency is the most common and the least deadly of the chronic valvular diseases. Whether or not minor lesions ever terminate in recovery may well be questioned. It is certain, however, that there are many cases in which full compensation is early established and maintained through life. The signs persist, but cardiac symptoms are absent. The outlook is more favorable in the cases which follow acute endocarditis than in those originating in sclerotic processes: when the disease begins in adolescence or early adult life than in childhood or the aged: in those who are able and willing to lead quiet, orderly, and well-disciplined lives than in those whose circumstances demand unremitting toil, or whose habits are irregular and self-indulgent. The extent of the leakage, as indicated by the degree of dilatation and hypertrophy, the completeness of compensation, the presence of secondary morbid conditions or of other valvular lesions or extensive pericardial adhesions has an important bearing upon the prognosis. The liability to recurrent acute endocarditis adds to the gravity of the condition. Among the common causes of death are cardiac insufficiency in the sense of loss of contractile power, pulmonary infarct, cerebral embolism, nephritis, and acute intercurrent disease.

iv. Mitral Stenosis.

The mitral orifice is narrowed or constricted and the passage of the blood from the left auricle to the left ventricle is impeded. The lesion is commonly the result of acute endocarditis in early life. It consists of thickening and contraction of the segments of the mitral valve or the ring, or both. Anatomically there are various forms. The more important are, (a) great thickening of the valves with a mere oblong fissure or chink—*buttonhole contraction.* The ring is often much contracted. (b) The projection from the line of the base of the segments of thick nodular fibroid masses, often in part calcareous, into the lumen of the orifice. The segments themselves may be thickened but are sometimes but slightly affected. (c) The valves are adherent at their borders but thin and elongated, projecting into the ventricle and opening at the tip by a constricted orifice—*funnel-shaped stenosis.* (d) Narrowing of the mitral ring without marked changes in the valve—probably a congenital condition. The chordæ tendineæ are shortened and thickened, and in some instances the tips of the papillary muscles are inserted into the deformed valves.

The degree of stenosis varies from the tip of the finger to an opening that will only admit a medium-sized Bowman's probe. The heart is moderately enlarged, the hypertrophy affecting the left auricle and right ventricle. The left ventricle, except in cases in which there is also marked insufficiency of the valve, is usually small. The derangement of function consists in the overfilling of the auricle with increase in its work during the ventricular diastole. The wall of the auricle undergoes hypertrophy, which for a time may compensate the defect. The pressure is transferred through the pulmonary circuit to the right ventricle, upon which the compensation largely falls, and which at first undergoes hypertrophy without dilatation. When compensation fails, there is relative incompetency of the tricuspid valve and transference of pressure to the venous side of the general circulation. In consequence of the inability of the left auricle to maintain compensation, as a rule, and the action of the hypertrophied right ventricle through the pulmonary circuit, the compensation in mitral stenosis is rarely complete.

Etiology.—Predisposing Influences.—Uncomplicated mitral stenosis is a rare affection. In almost all the cases there is some degree of mitral insufficiency. Age is important. A few of the cases are congenital; the greater number occur in early life. The evidences of the lesion may first attract attention at any period of life. There is a remarkable preponderance of cases in females. The ratio varies according to various statistics from 2 to even 4 to 1. This disparity of incidence affects only the cases in persons under middle age in whom the lesions are commonly due to endocarditis, and does not appear in the statistics of the old cases, which are almost always of sclerotic origin.

Exciting Cause.—Acute endocarditis in the course of an attack of rheumatism, chorea, scarlet fever, or one of the other acute infections of childhood may be the starting-point of mitral stenosis. The acute and repeated tension upon the heart valves in whooping-cough may act in the same way. In a remarkably large proportion of the cases the anamnesis is silent as to the cause.

Symptoms.—Fair compensation is often maintained for years, during which symptoms are absent. As it gradually fails dyspnœa upon exertion first attracts attention. Pressure paralysis of the left recurrent laryngeal nerve from the enlarged auricle has been observed. Cerebral embolism is by no means rare. The frequency with which hæmoptysis occurs while compensation is yet fair is interesting. The cases are often mistaken for incipient phthisis. The tendency to dropsy is less marked than in mitral insufficiency.

Physical Signs.—Upon inspection there is very often, especially in the young, precordial prominence due to hypertrophy of the right ventricle. The apex beat is commonly displaced but slightly toward the left, and may be indistinct, the visible impulse being at the lower end of the sternum and extending to the left costal cartilages. The pulsation of the conus arteriosus may often be visible at the sternal end of the third and fourth left interspaces. As compensation fails, the impulse loses its power, and signs of back pressure in the systemic veins appear, as distention of the superficial veins, especially the jugulars, with pulsation due to contraction of the right ventricle and enlargement of the liver. Upon palpation there is recognized in at least three-fourths of the cases a very distinct thrill. This sign is usually coarse in character, diastolic or presystolic in time, confined to an area above the apex, usually in the fourth and fifth interspaces, and circumscribed. It is more intense and slightly more extended during expiration and runs up to a short, sharp apex beat. This

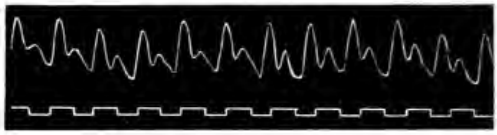


FIG. 352*A*.—Mitral stenosis; carotid tracing.

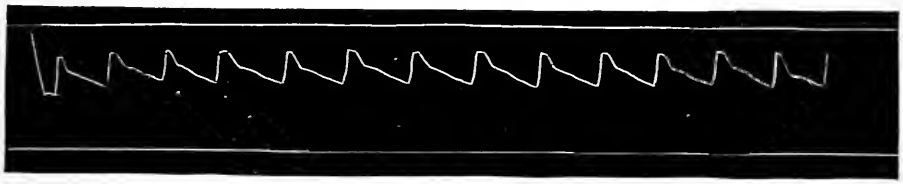


FIG. 352*B*.—Mitral regurgitation and stenosis; radial tracing.

thrill is of the highest diagnostic significance. It is the tactile equivalent of the characteristic murmur of mitral stenosis. It may, however, frequently be felt when no murmur can be detected at this time in the cardiac cycle. In stenosis of moderate grade, so long as compensation is good the pulse has no special characters. Upon failure of compensation it becomes small, soft, and arrhythmic. Upon percussion the dulness in fair compensation extends but slightly to the left of the normal line, rarely much beyond the midclavicular line, and little to the right of its normal limits. When, however, compensation is broken, the transverse diameter of deep cardiac dulness is decidedly increased, reaching beyond the midclavicular line on the left and beyond the parasternal line on the right. Auscultation reveals a characteristic murmur which corresponds in situation and in time of its occurrence in the revolution of the heart to the thrill which has been described above, and which is its auditory equivalent. This mur-

mur is rough and vibratile in character, increasing in intensity toward its close, and terminates abruptly in the tap which constitutes the first sound. It occurs in four modifications. In the greater number of cases it occupies the entire period of the diastole, and is increased in intensity at the time of the auricular contraction which immediately precedes the ventricular systole—the *presystolic murmur* of mitral stenosis; it may be heard at the beginning and at the close of the diastole—*diastolic rumble;* it may be present in the mid-diastolic period; and finally it is in some cases heard only in the moment immediately preceding the first sound. The intensity of this murmur is extremely variable. It may be the loudest of heart murmurs, or very soft and faint, or, finally, it may be wholly inaudible when the patient is at rest, and only heard after exertion. It may at times have a peculiar rumbling quality. Its loudness depends in part upon the character of the lesions and in part upon the force of the blood stream. It is in accordance with this fact that, as compensation fails, the murmur can no longer be heard, but only the sharp first sound in the mitral area, or the *one, two, three* of the gallop rhythm. If, however, under rest and treatment compensation is for a time re-established the murmur reappears. This change is often met with in actual practice and has been the cause of many disputes as to diagnosis.

The first sound at the apex is short, valvular, and very loud. It has in many cases the character of the second sound and is frequently mistaken for it. Under this error the murmur is naturally assumed to be systolic and the condition that of mitral insufficiency. This mistake is to be avoided by determining the time of the auscultatory signs by palpation of the cardiac impulse or carotid pulse at the moment of auscultation. The alteration in the first sound is due to the quick contraction of the left ventricle upon an abnormally small blood content. The pulmonary second sound is strongly accentuated. The electrocardiogram shows a high P-wave, when the auricle is hypertrophied.

Diagnosis.—The DIRECT DIAGNOSIS is in well-marked cases not difficult. It rests upon the association of the presystolic thrill and murmur with the signs of hypertrophy of the right ventricle, the absence of signs of enlargement of the left ventricle, the loud snapping character of the first sound, and the accentuation of the pulmonary second sound.

Prognosis.—In general the outlook is less favorable than in mitral insufficiency. This form of chronic valvular disease is only second to aortic insufficiency in the gravity of the prognosis. Sudden death is somewhat more frequent than in mitral insufficiency, but occurs usually after the compensation has become greatly impaired. Among the more common causes of death are progressive cardiac asthenia, pulmonary infarction or œdema, and acute intercurrent disease. More than any other chronic valvular disease of the heart, mitral stenosis is associated with tuberculosis, and to the latter the termination must in many of the cases be attributed.

v. Pulmonary Insufficiency and Stenosis.

Lesions of the pulmonary valves are extremely rare. Murmurs having their point of maximum intensity in the pulmonary area are common. They are usually systolic. They are sometimes present in health, especially in children, and are best heard during expiration and in the recumbent

posture, in the rapidly acting heart, in anæmia and chlorosis; and it is in the last that cardiorespiratory murmurs are usually heard.

Insufficiency.—This is a rare congenital lesion. It may occur in malignant endocarditis. Relative insufficiency may result from overdistention of the pulmonary artery. There are dilatation and hypertrophy of the right ventricle, epigastric pulsation, a heaving impulse over the lower sternal region, and a soft diastolic murmur at the second left costal cartilage and third intercostal space, much more distinct upon expiration. This condition is to be differentiated from aortic insufficiency, to which it has superficial resemblances, by the presence of epigastric pulsation and other signs of hypertrophy of the right ventricle, and the condition of the systemic arteries.

Stenosis.—This practically occurs only as a congenital lesion and is always associated with other developmental anomalies "in blue babies." Sclerotic changes occur and the deformed valves are especially disposed to acute endocarditis. There are no special symptoms. Among the physical signs are a systolic murmur and thrill in the second left intercostal space at the sternal border, a faint or inaudible second sound, and the evidences of hypertrophy of the right ventricle. The pressure of an aortic aneurism may narrow the pulmonary outlet, or the cicatrices of syphilitic lesions, the conus arteriosus, and give rise to similar signs. The diagnosis may sometimes be made with precision.

vi. Tricuspid Insufficiency and Stenosis.

Insufficiency.—This valvular defect is extremely rare as the result of endocarditis involving the right side of the heart. Relative insufficiency is, however, very common as a secondary condition in the period of failing compensation in disease of the aortic and mitral valves, especially the latter. It occurs also in advanced fibroid phthisis, emphysema, and other pulmonary diseases in which there is permanent obstruction of the pulmonary circulation. The auricle is dilated and hypertrophied. The return of the venous blood is impeded and the supply to the pulmonary artery diminished. The symptoms of retarded pulmonary circulation and visceral congestions characterize this condition. The physical signs are chiefly due to the enlargement of the right auricle and the reflux of blood from the right ventricle through the tricuspid orifice. They consist of increase in the transverse dulness to the right of the sternum with occasional pulsation at the sternal ends of the lower interspaces; a systolic murmur, usually soft and low-pitched, having its point of maximum intensity over the lower part of the sternum and propagated in the direction of the right axilla; and finally pulse-waves in the veins of the neck, more

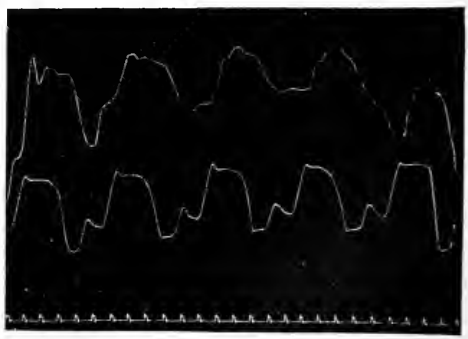


Fig. 353.—Venous pulse of tricuspid insufficiency. Upper tracing—venous pulse; lower tracing—apex beat.

marked upon the right side. The venous pulse is synchronous with the carotid pulse and apex beat. It is sometimes transmitted to the subclavian and axillary veins. It may, by way of the vena cava and hepatic veins, reach the liver and cause the phenomenon known as pulsating liver, an expansile pulsation in the organ best appreciated upon bimanual palpation, and to be differentiated from the "jogging" liver, which rises and sinks under the influence of the movements of the heart or aorta but does not expand and contract.

Stenosis.—Tricuspid stenosis is a rare form of valvular disease. It may be congenital, in which case it is almost invariably associated with other cardiac anomalies; or acquired, when it presents deformities similar to those seen in mitral stenosis. It is usually associated with some degree of incompetence of the tricuspidalis and with diseases of the other valve

vii. Physical Signs of Uncombined Valvular Lesions of the Left Heart, Compensation Being Maintained.

	Mitral		Aortic	
	Insufficiency	Stenosis	Insufficiency	Stenosis
Impulse.....	Increased in extent. Displaced to left	Often ill-defined and extended, but not much beyond the midclavicular line. Preceded by thrill	Extended, heaving, forcible and displaced downward and to the left	Variable; sometimes feeble and indistinct; often slow, heaving and forcible. Moderately displaced to the left.
Cardiac dulness	Increased transversely and downward. It may extend beyond the right border of the sternum and beyond the left midclavicular line	Increased to the right of the sternum and along its left border; not usually beyond the midclavicular line	Increased to a greater extent than in any other valvular lesion. Principally downward and to the left	Moderately increased to the left.
Murmurs.....	Systolic at apex accompanying or replacing the first sound. Transmitted toward left axilla. May be heard at the back; sometimes widely over chest	Coarse, presystolic, terminating in the first sound and limited to the region of the apex	Diastolic, prolonged, accompanying or wholly replacing the aortic second sound, and propagated from the base of the heart downward along the sternum and to the left	Coarse, systolic, of maximum intensity at aortic cartilage and propagated into the great vessels.
Associated signs	Accentuation of pulmonary second sound	Presystolic thrill; impulse of conus arteriosus of right ventricle in fourth and third, sometimes in second left interspaces. A clear, well-defined first sound. Accentuation of pulmonary second sound. Reduplication of second sound	A systolic shock in the larger arteries and sometimes a double murmur—Duroziez's sign. The aortic second sound may be heard over the right carotid when absent in aortic area	Systolic thrill in aortic area, absent or feeble aortic second sound. Occasionally a diastolic murmur.
Pulse.....	Full, regular, frequent and usually of low tension. Upon failure of compensation there is usually arrhythmia which is commonly persistent	Smaller in volume than normal and frequently arrhythmic	Water-hammer, collapsing—Corrigan's pulse	Small, of fair tension, regular, and usually somewhat slower than normal.

systems, most commonly the mitral. In a large proportion of the cases there has been a history of rheumatic fever; in a few a history of syphilis. This form of chronic valvular disease is far more common in females than in males. It has, in a majority of the cases, been first observed early in adult life. It is so rarely an isolated lesion that its symptoms are commonly obscured by those of the associated affections. They are those of general venous engorgement. Distention of the jugulars, cyanosis of the lips and face, tenderness over the liver, and dropsy are common. The physical signs are also usually subordinate to those of the associated valvular disease. Percussion shows increased dulness to the right of the sternum. When, in a case of mitral stenosis, there is a second presystolic murmur best heard at the base of the ensiform cartilage at the right, associated with a presystolic thrill, and these are of different character from those in the mitral area, tricuspid stenosis may be diagnosticated. The clinical diagnosis is, however, uncertain. Almost all the cases have been discovered upon the post-mortem table, the condition not having been suspected during life.

viii. Combined Valvular Diseases.

There are two cardinal facts in regard to chronic valvular disease. First, that the lesions are very rarely uncomplicated, and second, that the condition of the myocardium is much more important than the state of the valves. While the recognition of these facts renders heart disease complex and difficult as regards diagnosis, it greatly simplifies it in respect of prognosis and the recognition of the indications for treatment. The valvular damage is irreparable, but injury to the myocardium may be postponed or to some extent repaired. The diagnosis, to be of service, must be at once anatomical and functional. It undertakes to determine what valve system is defective; whether the defect gives rise to obstruction or incompetence or both, and in the last of these conditions which defect predominates; whether more than one valve system is involved, and in what manner; and finally the condition of the myocardium—is its contractile function increased to the point of full compensation? Is the increase maintained? Is it failing? From this point of view the combined lesions acquire especial clinical importance. In aortic and mitral insufficiency resulting from acute endocarditis a progressive stenosis may favorably modify the condition of the heart by diminishing the regurgitation. Aortic lesions and, in particular, aortic regurgitation are more frequently simple than mitral lesions. Relative and muscular mitral and tricuspid insufficiency are frequently uncombined.

Especially common is the combination of mitral insufficiency with stenosis of the mitral orifice. The greater the one, the less the other of these defects. This combination is very often associated with other valvular lesions. Next in order of frequency is the association of aortic insufficiency and mitral insufficiency, or combined mitral insufficiency and stenosis. Combined aortic insufficiency and stenosis is less common but not rarely associated with other valvular lesions. In proportion as the stenosis is marked the insufficiency is diminished and a progressive narrowing of the orifice in insufficiency may be conservative.

The frequent association of tricuspid disease with mitral lesions has already been noted, especially that of tricuspid and mitral stenosis. The diagnosis of these and other combined lesions depends upon the recognition of the associated signs and demands the painstaking study of every phenomenon in any particular case.

VII. CONGENITAL LESIONS OF THE HEART.

These lesions are of two kinds, developmental and inflammatory.

Developmental Defects.—To this group are to be referred the following anomalies:

Fig. 354.—Clubbed fingers and toes.—German Hospital.

(a) Defects of the whole heart, as acardia, double heart, ectopia cordis—conditions not, as a rule, of diagnostic interest. (b) Defects of the septa, which may, by the absence of both the auricular and ventricular septum, convert the heart into a double chamber—the bilocular heart; or by the absence of the ventricular septum only, convert it into a three-chambered viscus—the trilocular heart. (c) Patent foramen ovale. (d) Defects of the valves, which may be increased or diminished in number, or adherent. These anatomical defects involve the semilunar cusps of the aortic and pulmonary valves, but not the segments of the auriculoventricular valves. (e) Transposition of the large vessels. (f) Transposition of the heart—*Situs inversus cordis.* This developmental anomaly is usually associated with a general inversion of the viscera.

Fetal Endocarditis.—The right heart is usually affected. Pulmonary stenosis is a common result. Complete obliteration of the pulmonary orifice is associated with persistence of the ductus arteriosus and patulence of the foramen or other defect in the ventricular wall. Congenital lesions at the aortic orifice are rare.

Developmental anomalies are usually multiple. They not infrequently are the seat of sclerotic processes.

Symptoms.—Cyanosis, general duskiness of the surface, a persistent low external temperature, dyspnœa and cough, increase in the red blood-corpuscles, the drum-stick deformity of the fingers and toes, and finally

retarded physical and mental development are usually present. Associated developmental defects in the mouth, genitalia, or elsewhere are common. In infants the presence of endocardial murmurs with or without enlargement of the heart constitutes an important diagnostic criterion.

Diagnosis.—The diagnosis rests upon the association of several or all of the above conditions in a child or young adult in whose case there is a history of having been a "blue baby," or having had "the blue disease." The differential diagnosis of the various congenital defects must be determined by a special study of the physical signs in individual cases. In many of the cases a positive diagnosis of the particular lesions *intra vitam* cannot be made.

DISEASES OF THE ARTERIES.

I. ARTERIOSCLEROSIS.

Definition.—A disease of the arterial system characterized anatomically by degenerative changes in the media and adventitia with compensatory sclerotic connective tissue thickening in the intima, which thickening subsequently involves all the coats; and clinically by functional derangements in the various viscera.

The anatomical changes may be diffuse or localized. In the diffuse variety the arteries are dilated and tortuous, their walls thickened and inelastic, and the intima, the seat of irregular thickening, calcareous plates, and atheromatous ulcers; in the localized or circumscribed form yellowish-white, rounded, nodular patches, raised above the surface, are irregularly scattered along the intima. Diffuse and nodular sclerosis are commonly associated. Arteriosclerosis is the most common and varied affection of the arterial system. It is essentially progressive in character, though there are cases which beginning early in life run a very prolonged course without giving rise to conspicuous symptoms. More commonly the disease develops at a later period and is accompanied by characteristic changes in the visible superficial arteries and the evidences of lesions of the viscera and nervous system.

The degenerative and sclerotic processes show remarkable variations in correspondence in different cases and at different points. When degeneration is more active than sclerosis the arterial wall loses its tone, becomes diffusely dilated and may rupture. This is especially the case in the small cerebral arteries—miliary aneurisms, apoplexy. Atheromatous ulcers may occur, with the formation of emboli or thrombi, or partial rupture of the wall of a large artery may permit the blood to pass between the coats of the vessel and form a dissecting aneurism. When on the other hand the sclerotic process is more active the vessel walls become thickened at the expense of the lumen and there is retardation of the blood-flow, arterial hypertension and stress upon the heart and great vessels. An obliterating endarteritis, with local thrombosis and deposition of lime salts may result in cerebral softening, circumscribed myomalacia with rupture of the heart wall, coronary sclerosis with angina, or gangrene of the extremities.

In senile arteriosclerosis the arteries are dilated and tortuous, the walls thin and rigid, and the subendothelial tissue the seat of circumscribed collections of softened or broken-down material—*atheromatous abscesses.* When these collections rupture into the lumen of the artery they give rise to atheromatous ulcers.

In the various forms the degeneration of the media may be marked in the smaller arteries. The capillaries are thickened and may be obliterated. The connective-tissue overgrowth leads to more or less complete atrophy of the muscular and connective-tissue elements. In some cases characterized by general or local increase in blood-pressure the muscular fibres of the media may be preserved or even hypertrophied.

In consequence of these changes the larger and middle-sized arteries are dilated; the smaller, by reason of the thickening of the intima, are narrowed and very often wholly obliterated—*endarteritis obliterans.*

The ramifications of certain arteries are involved with greater frequency than others. In the series of cases studied by pupils of Thoma and analyzed by Bergmann, sclerotic changes were found in the ulnar artery in 94 per cent., the tibialis antica 93, the subclavian 88, arteries of the brain 87, internal carotid 87, radial 86, splenic 82, popliteal 79, external carotid 78, axillary 71, femoral 69, common carotid 68, ascending aorta 67, abdominal aorta 64, external iliac 58, and brachial 55. The minute arteries of the various organs are involved in the arteriosclerotic process. These lesions are especially common in the heart, brain, kidneys, liver, and pancreas. Sclerosis of the pulmonary artery and its branches is a constant concomitant of lesions such as chronic disease of the mitral valve, emphysema, and fibroid phthisis, which cause persistent increase of the blood-pressure in the pulmonary circulation. The artery may be dilated, with insufficiency of the semilunar valve system, its primary and secondary branches the seat of aneurismal dilatation, and its smaller branches highly sclerotic. The sclerotic process frequently extends to the capillaries and may also affect the veins, which not infrequently are sclerotic in the absence of similar changes in the arteries.

Etiology.—The following factors are of importance in the causation of arteriosclerosis:

1. *Persistent High Blood-pressure and Sudden, Frequent, and Extreme Alternations of Pressure.*—There are many conditions which bring about habitual strain upon the arteries. Among the more important are: (a) Habitual hard work. This accounts for the great frequency of arteriosclerosis among the laboring classes, and the fact that working men more frequently suffer from sclerotic changes in the upper extremities and women in the same walk of life in the lower extremities, while such changes in the arteries of the extremities are infrequent in persons whose occupations are not laborious (Romberg). The muscular effort habitually increases the peripheral resistance and raises the intra-arterial pressure. (b) Nervous influences—the strenuous life in which physical effort, mental stress, and excitement combine to tax alike the brain and the heart. In this connection the frequency with which arteriosclerosis is present in neurasthenia is to be considered. (c) Obesity. The increased effort demanded by the ordinary movements of life and by the larger volume of the circulating

blood tends to arteriosclerosis, and especially to arteriosclerosis of the coronary arteries. (d) Chronic interstitial nephritis is associated with persistent increase of blood-pressure and sclerotic changes in the arterial walls. (e) Frequently repeated and extreme changes in temperature, such as are necessary in certain crafts, tend by the abrupt contraction and dilatation of the superficial vessels to the production of arteriosclerosis. (f) The strain upon the arteries in aortic insufficiency rapidly brings about sclerosis of their walls.

2. *Chronic Intoxications.*—(a) The abuse of tea, coffee, tobacco, and alcohol is credited with a causal influence which is doubtless over-estimated. These narcotics, and especially alcohol, may exert an indirect effect by increasing nervous excitability. (b) Lead, diabetes, and gout play an important rôle. The mode of action has not been explained. Their association with interstitial nephritis is to be considered. (c) Renal disease. There are two groups of cases, primary interstitial nephritis and nephritis associated with general arteriosclerosis. (d) The infectious diseases, especially malaria, rheumatic fever, and enteric fever, appear in some cases to be the starting-point of progressive arteriosclerotic changes. (e) Excesses at table. There can be no doubt of the importance of over-eating as a factor in the causation of arteriosclerosis, and that the results of such excesses belong in the category of the chronic intoxications is equally beyond question.

3. *Syphilis* is a causal factor of great moment. In syphilitic subjects arteriosclerosis develops early and attains a high grade. The distinction between arteriosclerosis and the specific vascular lesions of syphilis is to be made.

4. *Heredity.*—The predisposition to arteriosclerosis varies greatly in different families. Inherited anatomical peculiarities, as congenital narrowness of the arteries and thickness of their walls, are attended with the liability to early sclerosis. These arterial changes appear early among the peasant immigrants from Italy and other countries of Southern Europe.

5. *Age.*—The view is generally entertained that arteriosclerosis is a manifestation of senile involution. There are, however, aged persons in whom little or no evidence of the disease is apparent. It is probable that in many cases the arteriosclerosis of old persons is a late result of the action of other causes operative in earlier life. In fact several of the above etiological factors are usually to be recognized in the anamnesis. This is especially the case in connection with syphilis.

It is in accordance with the above facts that arteriosclerosis is more common after middle life than at an earlier period, and in men than in women.

Symptoms.—Arteriosclerosis begins insidiously and may long remain latent. There are, however, cases in which the vascular changes attain a high grade in the course of a very few years. When symptoms become manifest they may be general or local, according to the vascular distribution chiefly affected.

The general symptoms are in many cases not different from those of old age. The skin becomes harsh, wrinkled, and inelastic; subcutaneous fat is reduced over the chest and extremities and accumulates upon the

abdomen; the muscles waste; the viscera undergo atrophic changes; and there is manifest loss of bodily and mental power. Dyspeptic symptoms are often prominent. Characteristic phenomena relate to the circulation. The blood-pressure is increased, the superficial arteries are thickened and hard, there is hypertrophy of the left ventricle and accentuation of the aortic second sound, which has often a clear, ringing quality. There are, however, cases in which the blood-pressure remains low and the hypertrophy of the left ventricle is moderate. An increased flow of urine of low specific gravity and transient traces of albumin, together with occasional hyaline casts, is not uncommon.

The local symptoms depend upon the grade of the vascular lesions and the organ or structure principally involved, as the heart, brain, kidneys, or extremities.

1. Cardiac Symptoms and Signs.—The effect of persistent resistance to the flow of the blood in the peripheral arteries is hypertrophy of the left ventricle, with the symptoms and physical signs of that condition. When the coronary arteries are involved, local or general myocardial degenerations occur with their attendant symptoms, which are those of cardiac insufficiency. Angina pectoris, aneurism of the heart, local softening, rupture, or sudden death may result. When dilatation supervenes the systolic apex murmur of relative mitral insufficiency is heard, and the symptoms of extreme cardiac insufficiency arise, dyspnœa while at rest, somnolence, scanty urine, dropsies and effusions into the serous sacs. Dilatation of the ascending aorta may give rise to tympany or dulness at the sternal end of the second right intercostal space or palpable pulsation in the episternal notch and a soft systolic murmur with an accentuated second aortic sound when the aortic valves are capable of closure, and may be well shown by the Röntgen rays.

2. Cerebral Symptoms.—Among the early symptoms are sensations of fulness and pressure in the head, insomnia, distress, anxiety, and vertiginous attacks. As the vascular lesions progress mental symptoms develop. The attention flags, the recollection of recent events fails, the patient becomes indifferent about his personal appearance and less considerate of others. Self-restraint may be impaired. Actual vertigo, transient loss of consciousness, and temporary derangements of speech are among the symptoms of advanced arteriosclerosis. Repeated attacks of hemiplegia, monoplegia, and aphasia may occur with the signs of organic lesions and terminate in complete recovery in the course of a few hours or a day or two. The Stokes-Adams syndrome is occasionally observed. Tinnitus cerebri and tinnitus aurium are common and often distressing symptoms. The cerebral symptoms of arteriosclerosis include those of the lesions of the brain due to persistent obstruction of the smaller vessels, and accidents, such as thrombosis, embolism, and hemorrhage.

3. Renal Symptoms.—These correspond to those of chronic interstitial nephritis. The association of this form of renal disease and arteriosclerosis is common. Arteriosclerosis predisposes to nephritis; chronic nephritis to general sclerosis. When the patient has not been under observation from the beginning it is impossible to determine which is the primary, which the secondary, affection. In either case the renal symptoms may

dominate the clinical picture; more commonly, however, the cardinal symptoms are those of myocardial disease. The condition constitutes one of the forms of cardiorenal disease. Glycosuria is a frequent indication of sclerosis of the pancreatic arteries.

4. Vasomotor Symptoms.—Sensations of cold and numbness in the hands and feet, fulness or lightness in the head, tingling and pulsation in the fingers, dynamic pulsation in the abdominal aorta, and congestion of the feet and legs are very common.

5. The Extremities.—The symptoms due to sclerosis of the peripheral vessels are comparatively infrequent. The diminished capillary circulation gives rise to more or less marked pallor, which is not rarely an early symptom. Its association with loss of weight and strength suggests anæmia or even the development of visceral cancer, especially in cases in which the signs of changes in the heart or brain are not prominent.

Intermittent Claudication; Dysbasia Angiosclerotica; Crural Angina.—This constitutes a somewhat common and very striking clinical manifestation of sclerosis in the arteries of the lower extremities. It depends upon the fact that while at rest the blood supply to the muscles, diminished as it is by the narrowing of the lumen of the peripheral vessels, is yet sufficient for their physiological requirements or even for moderate use, but when muscular effort is made the blood supply is inadequate and severe cramp-like contractions ensue. After walking a short distance the patient experiences in one or both legs sensations of numbness, tingling, heat or cold, tension and pain. The skin becomes pale and cyanotic. If the effort is not discontinued more or less severe cramp and muscular disability follow. These symptoms cease upon rest, only to return upon further effort. In extreme cases the spasms recur upon moderate effort and the muscles undergo gradual atrophy.

Diagnosis.—The direct diagnosis of arteriosclerosis may be made when increased blood-pressure, thickened and tortuous superficial arteries, hypertrophy of the left ventricle, and accentuation of the aortic second sound are present. The thickened radial artery can often be rolled under the finger upon the radius like a whip-cord, or it may show irregular, nodular projections along its course like a string of wampum, or present little aneurismal-like circumscribed dilatations. A high degree of sclerosis in the superficial arteries may exist without a corresponding condition in the arteries of the parenchymatous organs, and the converse is true. Another important fact in diagnosis is that advanced arteriosclerosis may involve a vascular territory or organ, as the kidneys or brain, without, in the absence of general sclerotic changes, increasing the blood-pressure, and, therefore, without causing left ventricle hypertrophy or marked accentuation of the aortic second sound. When there is marked deposition of lime salts in the affected arterial walls in the legs and feet, the X-ray examination yields positive results, but it is useless in the deeply seated arteries of the trunk, with the exception of the aorta, the shadow of which is broadened and intensified in places by the presence of thickened atheromatous plates. Etiological considerations are important in the diagnosis.

Prognosis.—The course of arteriosclerosis shows extreme variations. The disease may involve the peripheral circulation and be wholly latent as

to symptoms. Cases of this kind go on from year to year without serious impairment of health. It may, on the other hand, rapidly give rise to distinctive changes in organs that are necessary to life. Finally, it not uncommonly sets up a vascular dyscrasia in which the entire body and its organs are involved in progressive nutritional and atrophic changes, which terminate in death in the course of a few years. In selected cases of peripheral sclerosis the removal of the cause and regulation of the mode of life very often favorably influence the progress of the disease and may arrest it. In cases due to syphilis an energetic antiluetic treatment may be followed by an arrest of the sclerotic process.

II. ANEURISM.

Definition.—A circumscribed dilatation of an artery. This anatomical condition is to be distinguished from the diffuse widening of the larger arteries which occurs in arteriosclerosis.

Aneurisms are divided:

(a) According to their form into *fusiform* or *cylindroid,* in which there is uniform dilatation of the vessel, and *sacculated,* in which there is a lateral bulging or protrusion of the wall.

(b) According to the composition of the wall into *true,* in which the wall is composed of one or more of the coats of the artery, and *false,* in which there is a rupture of all the coats and the blood is confined by the surrounding tissues. This distinction is not important, since a false aneurism is in reality a hæmatoma and the differentiation between true and false aneurisms cannot in all cases be made after death, much less during life.

(c) According to other anatomical peculiarities into *cirsoid,* in which an artery and its branches are involved in the dilatation, *dissecting,* in which the blood collects between the coats of the artery—this type occurs in the aorta and occasionally forms a complete double tube—and *arteriovenous,* in which there is a communication between an artery and a vein. There may be a sac between the artery and vein, but more commonly the communication is direct and the vein which yields to the intra-arterial pressure is dilated, tortuous, and pulsating—*aneurismal varix.*

i. Aneurism of the Aorta.

Aneurisms of the aorta are usually fusiform or sacculated. The latter is more common. The combination of these forms is occasionally encountered. Dissecting aneurisms are rare. Still more rare are arteriovenous aneurisms. The essential anatomical condition is dilatation, under the intra-arterial pressure, of the vessel wall, weakened by disease. In fusiform dilatation all three coats of the vessel are dilated. In sacculated aneurism the intima may extend into the sac some distance; the media undergoes atrophic changes and extensive destruction. The wall of the sac is in the greater part of its extent formed by the adventitia, which is thickened, infiltrated, and fused with the surrounding tissues. The communication with the aorta is by an opening of variable size. The interior of the sac is lined by superimposed laminæ of coagulated blood, those

which are peripheral being dense and of a whitish color; those which are central being soft and red. The arrangement of these coagula is neither symmetrical nor constant, and important modifications of the pressure symptoms and physical signs arise in consequence of the yielding of the sac to the internal pressure in various directions at different periods in the course of the affection. In rare instances, small aneurisms having narrow communications with the lumen of the aorta are completely filled with coagula and thus undergo spontaneous obliteration with the formation of a small nodular tumor. As the conditions which give rise to sacculated aneurism involve different portions of the wall of the aorta multiple aneurisms are by no means rare. In size sacculated aneurisms vary from a walnut to a large cocoanut; in shape they are globular, but as they increase in size the wall yields more at one point than another in such a manner that irregular protrusions occur. Aneurisms may occur in any part of the aorta, from just above the ring to the iliac bifurcation. The most common site is in the ascending portion of the arch to the right.

Etiology.—Arteriosclerosis, syphilitic aortitis, and trauma are the most important etiological factors. The great majority of aortic aneurisms are due to those causes acting singly or in combination. Far less frequent are cases due to the action of micro-organisms.

(a) *Arteriosclerosis* leads to diffuse weakening of the wall, which yields to the pressure of the blood at a period when compensatory thickening of the intima has not yet occurred, and undergoes dilatation. Sacculated aneurism may occasionally have its origin in arteriosclerosis.

(b) *Syphilis* plays a most important rôle. Sacculated aneurism is in a large proportion of the cases syphilitic. Whether the process has its beginning comparatively early in the changes in the wall due to gumma or at a later period in consequence of the loss of elasticity in scar tissue remains undetermined. The lesions of a patch of syphilitic aortitis constitute the point of diminished resistance.

(c) *Traumatism.*—When the media and adventitia are weakened, a sudden increase of the blood-pressure may lacerate the intima. A violent contusion, a fall, a blow upon the chest may be followed by the gradual development of an aneurism.

(d) *Micro-organisms.* — Multiple aneurisms of the aorta and other arteries have occasionally been observed in connection with malignant endocarditis. Various bacteria have been found in the lesions. Weakening of the wall of the vessel in consequence of an ulcerative process analogous to that affecting the valves doubtless constitutes the early lesion.

(e) *Traction Aneurisms.*—An exceedingly rare form of aneurism is caused in the concavity of the aortic arch at the point of insertion of an insufficiently elongated remnant of the duct of Botallo.

Among other predisposing influences of secondary importance are the following: *Age.*—Aortic aneurism is much more common between the fortieth and sixtieth years than at any other period of life. *Sex.*—Men suffer more frequently than women in the proportion of 4 or 5 to 1, a preponderance due not to inherent anatomical differences but to the far greater exposure to the common causes. *Habits.*—The direct influence of alcohol has probably been over-rated. Its indirect influence in leading to exposure

to the danger of contracting syphilis is very great. *Occupations* which involve great muscular effort and those which are attended with the danger of violent blows and contusions of the chest may be regarded as predisposing to thoracic aneurism.

An aneurism of the aorta is a vascular tumor which may be wholly latent or manifest itself by symptoms, usually effects of pressure, and physical signs which differ according to its situation and the relative amount of stratified clot and blood which it contains. It is convenient to consider separately aneurism of the thoracic aorta and aneurism of the abdominal aorta.

(a) ANEURISM OF THE THORACIC AORTA.

Symptoms.—The relative prominence of subjective symptoms and physical signs depends upon the situation and size of the aneurism. The cases may be arranged in four groups.

1. The Aneurism is Latent.—Symptoms are wholly absent, or vague and not suggestive of the actual condition. There are no physical signs. This group includes the cases of moderate fusiform dilatation, small sacculated aneurisms in the ascending aorta, and especially those immediately above the sinuses of Valsalva. The last not rarely rupture into the pericardium. The diagnosis is made after death.

2. Symptoms are Prominent.—Pressure symptoms are present, but the nature of the lesion cannot be determined. To this category are to be referred small sacculated aneurisms of the transverse and descending portions of the arch compressing the trachea or left main bronchus and the recurrent laryngeal nerve, and larger sacs in various situations, containing much laminated clot and yielding the signs of a solid tumor rather than those of an aneurism, and sacs upon the descending aorta just above the diaphragm, especially those eroding the vertebræ.

3. Signs are Prominent.—There are associated subjective symptoms but they are distinctly subordinate to the objective manifestations of expansile pulsation, circumscribed dulness, thrill, diastolic shock, and tracheal tugging. This group comprises certain aneurisms of the convexity of the ascending portion of the arch which project to the right and exert moderate pressure chiefly upon the right lung, and some large aneurisms in this situation which have eroded the chest wall and formed projecting external tumors, with great relief from the pressure symptoms of an earlier period in the course of the disease. In both these sub-groups the contrast between the prominence of the physical signs and the moderate intensity of the symptoms may be very marked. Broadbent's division into the aneurism of symptoms and the aneurism of physical signs serves an important purpose in indicating the data upon which to base a diagnosis.

4. Symptoms are Severe and Signs are Distinctive.—Under this heading may be grouped a great majority of aneurisms of the ascending and transverse portions of the arch which have attained considerable size and are sufficiently free from coagula to constitute pulsating tumors. Both those still within the chest and those which have perforated the chest wall and form external masses belong to this group; also those aneurisms

of the descending aorta which compress the left lung or erode the vertebræ, and in particular those which erode the ribs and appear as external tumors at the back.

The connective-tissue overgrowth in the inflammatory capsule involves and compresses the nerve-trunks with which the tumor comes into contact. To this fact is to be attributed the pain so characteristic of growing aneurismal tumors, the palsies of the recurrent laryngeals and pneumogastric, and derangements of the sympathetic.

When neighboring blood-vessels are implicated in the growth, to which the venous trunks and the pulmonary artery are particularly liable, they are narrowed and the veins may be completely compressed, or per-

A

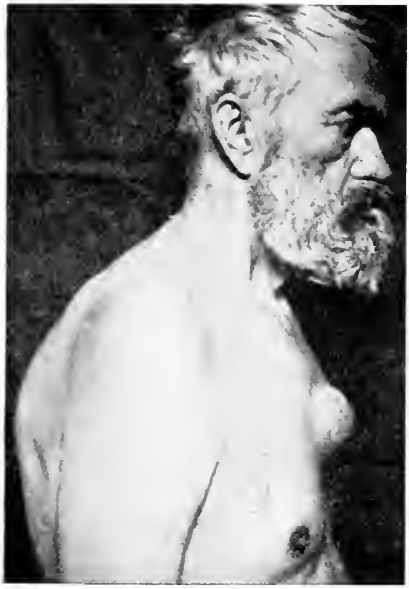


FIG. 355*A*.—Aneurism of the arch of the aorta protruding through the sternum.—German Hospital.

B

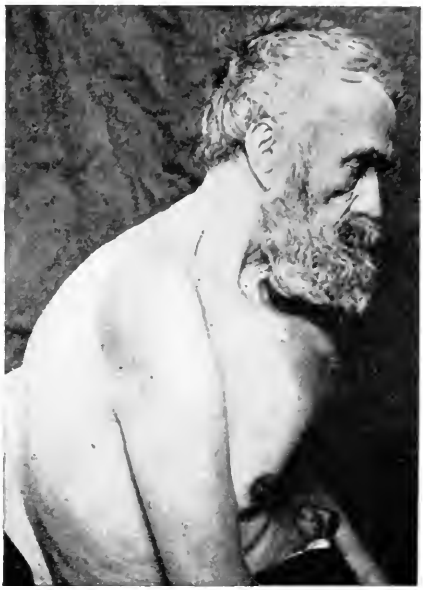


FIG. 355*B*.—Aneurism of the arch of the aorta protruding through the sternum. The same patient one year later. Death occurred suddenly from external rupture through the skin.

foration into the superior vena cava may take place. In a similar manner the trachea, a main bronchus, or the œsophagus may be involved and compressed, or the sac may rupture into these organs.

The sternum, costal cartilages, ribs, and the bodies of the vertebræ undergo more or less extensive erosion under the pressure of the distending sac. In rare instances portions of the bony wall of the chest are enclosed in the advancing sac, and a fragment of a rib or the end of a partly eroded clavicle is found within its capsule.

Hemorrhage.—The adventitia, even when reinforced by encapsulating connective tissue, may, in the course of a short time, yield to the blood-pressure. When the rupture takes place into the trachea, a bronchus, the œsophagus or stomach, the pleura or pericardium, death usually occurs at once. If the sac is surrounded by dense connective tissue a hæmatoma

is formed immediately at the seat of rupture and infiltrating the adjacent parts. Under these circumstances the bleeding may be arrested for a period, only, however, to recur from time to time until fatal hemorrhage ultimately takes place. The blood loss depends upon the amount of thrombus within the sac. If there is a considerable quantity of stratified clot, as is usually the case in aneurisms that have perforated the wall of the chest and ruptured externally, the bleeding may be moderate and for a time controllable. Recurrent hemorrhage frequently takes place in such cases for weeks or months. Protracted or recurrent small bleedings may occur under similar conditions into the trachea, bronchi, or œsophagus.

Fig. 356.—Aneurism of ascending aorta. Relief of pain upon appearance of external tumor, which occurred under observation.—Jefferson Hospital.

Pain is an early and important symptom. It may be in the sac itself,—*intrinsic*,—due to irritation of the sac or internal pressure. This pain is dull, aching, and substernal. More commonly it is in the adjacent parts,—*extrinsic*,—due to irritation of nerve-trunks implicated in the advancing capsule or subjected to pressure. This kind of pain is continuous, with paroxysmal exacerbations of great intensity, and is particularly severe at the time of erosion of the vertebræ or the wall of the chest. It is described as sharp, lancinating, cutting, boring, and the like. It often radiates along the intercostal nerves, or into the side of the neck and down the left arm. When the bodies of the vertebræ are eroded, the pain radiates in the course of the intercostal nerves and is often intense, suggesting herpes zoster or intercostal neuralgia. The pain of aneurism, usually spoken of as neuralgic, is in point of fact a symptom of pressure neuritis. When the aneurism is situated at the root of the aorta, attacks of angina pectoris of varying intensity may occur, with radiation to the left side of the neck and arm.

Cough is a common symptom. It may be caused by irritation pressure upon the vagus or recurrent laryngeal nerve, compression of the trachea or a main bronchus, in which event it is often associated with stridor, or by bronchitis. The expectoration is often blood-stained from interference with the venous circulation or from granulations at a point of impending rupture. In bronchitis and bronchiectasis it is thin and abundant. It is sometimes purulent and offensive.

Dyspnœa may be laryngeal, tracheal, or pulmonary, and these forms are sometimes present in the same case. Laryngeal dyspnœa is due to

irritation of the recurrent nerve and is usually associated with a ringing brassy cough, aphonia, and hoarseness. The tracheal form has its origin in direct compression of the windpipe or left primary bronchus and is accompanied by stridor; while pulmonary dyspnœa may be caused by compression of one or both lungs by an enormous aneurism of the lower or posterior wall of the transverse arch.

Dysphagia may arise when the sac develops in relation with the gullet. It may be due to œsophagismus or stenosis from compression. A clinical rule, in no case to be disregarded, is never to pass an œsophageal bougie if there is reason to suspect thoracic aneurism.

The signs of a thoracic aneurism are due, (a) to the tumor itself, and (b) to pressure upon adjacent structures.

Physical Signs.— Inspection. —In a large proportion of the early cases, and in many throughout the whole course of the disease, this method yields no physical signs. The tumor while still wholly intrathoracic may cause local bulging of the chest wall with widening of the intercostal spaces and visible pulsation. The signs may elude observation save in a good light and with oblique illumination. When perforation of the chest has occurred there is a more or less prominent external pulsating tumor, at the summit of which, in late cases, the skin may be livid or necrotic and the seat of hemorrhagic oozing. Such tumors are most common at the upper part of the sternum and to the right, and are sometimes of large size, extending also to the left. When the innominate is involved the pulsating prominence projects above the right clavicle and episternal notch. An aneurism of the descending aorta may give rise to a tumor to the left of the spine. Pressure upon venous trunks frequently causes congestion of the face and eyes, and enlargement of the superficial veins of the trunk and arm. These signs are much more common upon the right side. In consequence of pressure upon the sympathetic occasional differences in the pupils arise. Irritation of the upper dorsal or lower cervical ganglion upon the affected side is attended with dilatation of the pupil, which may or may not be accompanied by pallor of the face upon the same side; while destructive pressure is followed by contraction of the pupil and in some cases by flushing and sweating upon the same side of the face. The larynx may be seen to be displaced to the left, much more frequently to the right, by the pressure of an aneurismal tumor, and in large aneurisms of the arch tracheal

Fig. 357.— Aneurism of the aorta and innominate artery.

tugging may sometimes be seen. The visible apex beat is displaced downward and to the left, indicating more commonly dislocation of the heart from pressure than dislocation of the apex from hypertrophy.

Palpation.—This method confirms the signs obtained upon inspection. The extent, force, and expansile character of the pulsation are determined by this method of physical diagnosis. Intrathoracic aneurisms of considerable size may cause general expansion of the upper part of the thorax in its anteroposterior diameter, which is best recognized by bimanual palpation, one hand being firmly applied to the back, the other to the front of the chest. There may be merely a diffuse pulsation. An aneurism which has perforated the wall of the chest forms a distinct tumor with expansile pulsation, the denseness of which is determined by the amount of coagula. Very rarely the sac is thin-walled and fluctuating. There is usually a distinct diastolic shock, which may be intense and is of diagnostic significance. A systolic thrill may often be felt but is to be distinguished from the thrill of a dilated aorta and from the thrill which is present in aortic stenosis. Retardation of the pulse is common. The radials may be unequal in time and volume. When the sac is very large the pulse in the arterial trunks beyond it may be scarcely perceptible.

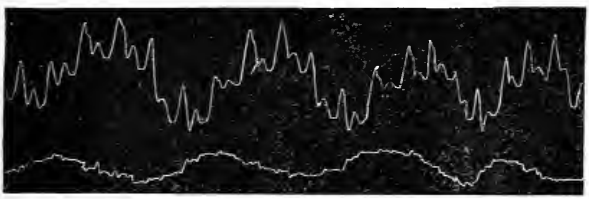


Fig. 358.—Upper tracing—carotid pulse in a case of aneurism of the arch of the aorta. Lower tracing—thrill over aneurismal tumor.

Tracheal tugging is common in aneurisms of the transverse arch and is a very important sign in deep-seated sacs not manifest by other signs. This phenomenon is not, however, pathognomonic of aortic aneurisms. It may occur in mediastinal tumors and in enlargement of the peribronchial lymph-nodes when so situated as to cause close adhesions between the aorta and the left primary bronchus.

Percussion.—Deeply situated aneurisms of small size yield no signs. The percussion signs of larger aneurisms which approach the chest wall depend upon the fact that they displace the lungs. The pulmonary resonance is replaced in aneurisms of the ascending limb of the arch by circumscribed flatness, usually to the right of the manubrium sterni; in aneurisms of the transverse arch by flatness in the upper sternal region to the left of the sternal border; and in those of the descending aorta by flatness along the left side of the spine and in the scapular region. The area of flatness corresponds to the region of contact between the tumor and the inner wall of the thorax, and is surrounded by a very narrow border of dulness and an outer border of tympanitic resonance, the width of which is determined by the extent to which the circumjacent lung is compressed.

Auscultation.—Murmurs may be absent even in aneurisms of large size. The greater the amount of stratified coagula the more nearly the aneurism resembles a solid mediastinal tumor. An accentuated second sound is a common and significant sign. A systolic murmur is often heard; less frequently a diastolic murmur, which is not a sign of the aneurism but

of an associated aortic insufficiency. An extremely rare continuous murmur with rhythmical whiffs corresponding to the systole heard over the manubrium is a sign of an arteriovenous aneurism or communication with the pulmonary artery. A systolic murmur may sometimes be heard over the trachea or at the open mouth of the patient.

The following clinical phenomena associated with aneurisms of different parts of the aorta are of diagnostic importance:

Aneurism of the Ascending Portion of the Arch.—Small sacs close to the root of the aorta are latent. Larger aneurisms arise most frequently from the convexity and develop toward the right. Deep-seated pain is an early symptom. It is most severe behind the manubrium and radiates to the neck, shoulders, and arms, more frequently the left than the right. Flatness in the sternal end of the second, third, and sometimes also the first intercostal space indicates the extension of the tumor to the right. In this region the heart sounds are loud and the second aortic sound is commonly ringing and accentuated. Expansile pulsation is present. The apex of the heart is displaced downward and to the left. Aneurisms of this part of the aorta may, in rare instances, communicate with the superior vena cava or compress the inferior vena cava, causing œdema of the lower extremities, and ascites. An aneurism springing from the concavity of the ascending portion may extend beyond the left sternal border. The right recurrent laryngeal nerve is exposed to pressure. Sclerosis of the coronary arteries, sclerotic lesions of the aortic cusps, and relative insufficiency are common. When a sacculated aneurism in this position attains considerable size, the overlying manubrium and cartilages and ribs to its right form a distinct, rounded, pulsating prominence, which in the course of time may cause absorption of the wall of the chest and appear as an external tumor. Systolic murmurs are common and are occasionally audible at a distance from the chest. Compression of the neighboring veins occurs early. The jugulars, especially the right, are prominent. The little venous twigs overlying the tumor are enlarged. The trachea and left bronchus are frequently compressed. Tracheal tugging may occur when firm adhesions with the trachea or bronchus are established. Difficulty in swallowing is not common in aneurisms in this situation.

FIG. 359.—Aneurism of ascending portion of the arch of the aorta.

Aneurism of the Transverse Portion of the Arch.—The sac may cause prominence of the manubrium and the cartilages and ribs to its right, as in aneurisms of the ascending aorta. The innominate is frequently involved, with the appearance of an external pulsating tumor at the sternoclavicular articulation and extending upward into the neck. Compression of the left recurrent laryngeal nerve is common. An early symptom is hoarseness with a curious high-pitched vibrating quality of the voice. In slight palsy

and in older cases the voice may be unchanged. Pupillary derangements are sometimes observed. Compression of the trachea and left bronchus, and tracheal tugging are very common. Dysphagia is more frequent than in aneurisms in other portions of the aorta. The sac sometimes includes portions of the ascending and transverse arch, causing dulness to the right and upward, and can often be felt pulsating in the episternal notch.

Sacculated aneurisms in the concavity of the arch are always difficult of recognition. Even when of moderate size they may cause persistent, even fatal, hæmoptysis. They elude physical examination and rarely attain a size sufficient to displace the heart. This form of aneurism may be suspected when, with persistent or recurring hæmoptysis in the absence of recognizable cause, are associated paralysis of the left recurrent nerve, dislocation of the trachea and larynx to the right, tracheal tugging, stridor, and dysphagia; but none of these is constant.

Fig. 360.—Unilateral clubbed fingers in aneurism of the descending arm of the aortic arch.—Groedel's case.

Aneurism of the Descending Arch.—These tumors may also be latent. Pain is a common symptom. It may be intrinsic. Very often, however, it is due to erosion of the dorsal vertebræ. Dyspnœa and stridor from compression of the left bronchus and lung, bronchitis, bronchiectasis, and bronchorrhœa may occur. Left-sided recurrent nerve palsy or paralysis, difficulty in deglutition, left pupillary phenomena are of diagnostic importance. When the sac is large, retardation of the crural pulse as compared with the radials or the apex beat is a less important sign. At the left sternal border in the first and second intercostal spaces there may be dulness, with distinct heart sounds, murmurs, and pulsation. Perforation may take place and an external tumor present in this region—an uncommon event.

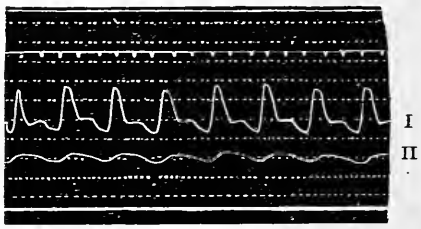


Fig. 361.—Sphygmograms in aneurism of the descending arm of aortic arch. I, right radial pulse; II, left radial. Same case as Fig. 360.—Groedel.

Aneurisms of the Descending Thoracic Aorta.—The sac is usually low down, resting upon the diaphragm and the left side of the bodies of the lower dorsal vertebræ, which are eroded. Among the pressure phenomena are dysphagia, intense pain radiating to the left, pleuritic friction, impaired resonance, together with feeble respiratory sounds and small mucous râles in the lower scapular region near the spine. A pulsating external tumor may present in the back. Pain is usually present, but the other phenomena may be wholly absent and the condition overlooked.

As a rule large aneurisms are sacculated and increase in size without involving other portions of the aorta than that from which they spring.

Exceptionally an aneurism arising from one portion enlarges by involving adjacent parts of the aorta until several parts are successively implicated, as the ascending portion, the transverse arch, and to some extent the descending arch. Under such circumstances the rational symptoms and physical signs of aneurism in the various localities are successively developed. Still more rarely multiple aneurisms are present. In the latter case one may be recognized by suggestive symptoms and characteristic signs, while the others may escape detection.

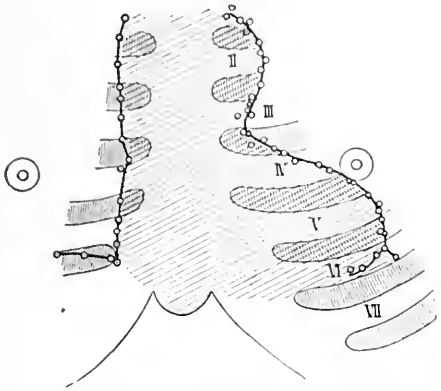


FIG. 362.—Orthodiagraphic outline of left border of aneurism of descending limb of arch of the aorta, showing displacement of the heart downward and to the left. Same case as Fig. 360.—Groedel.

Diagnosis. — DIRECT. — There are cases in which a positive diagnosis cannot be made. Aneurisms of the root of the aorta and of the concavity of the arch, when small, are usually wholly latent. The anamnesis is important. Syphilis, occupation, strain, blows and contusions of the chest are highly suggestive. Alcoholism is of secondary importance. Middle age and the male sex are predisposing influences of weight. Among the symptoms those which arise from intrathoracic pressure are significant. These comprise pain, dyspnœa, stridor, cough, dysphagia, pupillary differences, and a peculiar hoarseness with a high-pitched, shrill, vibrating quality of the voice. Among physical signs which point strongly to aneurism are, in the areas named, circumscribed flatness shading off to dulness, prominence with pulsation, a systolic murmur, systolic thrill, diastolic shock, displacement of the apex downward and to the left (especially when the signs of cardiac enlargement are absent), enlargement of superficial veins, inequality of radial pulses, a retardation or absence of crural pulsation and tracheal tugging. None of these clinical phenomena is diagnostic. The association of all of them is conclusive. When several of them are present the diagnosis becomes probable. An external tumor with distinctly expansile pulsation and diastolic shock justifies a positive diagnosis. Two facts are to be borne in mind. First, an enlarged lymph-node lying directly over a large vessel may pulsate synchronously with the action of the heart, but the pulsation is not expansile; and second, tracheal tugging may be present when, in mediastinal tumor or enlarged peribronchial lymph-nodes, there are close adhesions between the aorta and the left bronchus. Tracheal tugging is, therefore, not a pathognomonic sign of aneurism. The X-rays are of great value as showing, upon fluoroscopic

FIG. 363.—Aneurism of the descending thoracic aorta.

examination, a shadow in an abnormal situation, the borders of which expand and contract with the diastole and systole of the heart. Such a shadow is to be differentiated from the non-expansile movement which attends the advance and retreat of a tumor moved by the pulsation of the aorta or heart. The shadow of an aneurism attached to the trachea or left bronchus may rise with the act of deglutition, while tumors, in consequence of their firm attachments to the surrounding structures, are not affected by swallowing.

The situation of an aneurism may be determined by the foregoing symptoms and signs. But there are exceptions to this rule. Thus a sac springing from the ascending portion of the arch may present at the left border of the arch, while one connected with the descending portion may extend to the right of the manubrium and cause pulsation in the right interscapular space; and the possibility that an elongated aneurism of the ascending portion may cause dulness and pulsation to the right of the lower part of the sternum must be considered.

Differential.—Sclerosis of the aorta can scarcely be differentiated from fusiform aneurismal dilatation of the aorta. The fluoroscopic shadow is circumscribed in saccular aneurisms; diffuse and uniform in arteriosclerosis and the dilatation which occurs in functional derangements, as some cases of neurasthenia and exophthalmic goitre. Pain, which is an early and continuing symptom in aneurism, is not a prominent symptom in sclerosis. *Dynamic Pulsation.*—Increased dulness at the level of the upper part of the manubrium and pulsation in the episternal notch are sometimes present in cases in which, at the autopsy, no dilatation of the aorta is found. This form of widening of the aorta, if persistent, cannot, during life, be differentiated from fusiform aneurism. *Dislocation of the Aorta in Spinal Curvature.*—The convex border of the ascending limb of the arch may be displaced in such a manner as to cause dulness and forcible pulsation beyond the right border of the sternum and simulate an aneurism. Pressure symptoms and pain are usually absent, the pulsation is not expansile, and there is no diastolic shock. *Solid Intrathoracic Tumors.*—These are very often malignant. They take origin in the mediastinal lymph-nodes, the pleura or lungs, the thyroid body, or the œsophagus. A persistent thymus may be greatly enlarged. Such new growths commonly show a tendency to develop both to the right and left of the sternal borders, and not, as is usually the case in aneurism, upon one side. The heart sounds are not so loud as in aneurism, and diastolic shock is not felt. Murmurs may occur but are far less common. Differences in the radial and retardation in the crural pulse are likewise absent. Glandular metastases may be present in the neck—a sign of great significance. Pulsation is common but not expansile. Symptoms of pressure upon the recurrent laryngeal nerves are common, but other pressure symptoms are less prominent than in aneurism. The outline of an aneurismal sac is rounded and usually regular; that of a tumor uneven and irregular. The course of malignant disease is rapid, emaciation pronounced, and cachexia early developed; that of aneurism relatively slow, and the fatal issue may occur while the general nutrition is yet fair. There may be the history of a primary growth which has been removed, or the indications of its presence in a distant organ or part. Thoracic tumors and aneurisms containing

much stratified clot cannot always, when deep seated, be differentiated. *New Growths Involving the Wall of the Chest.*—Osteosarcoma may involve the sternum or ribs. The overlying veins are greatly enlarged and tortuous. The osseous structures are palpably enlarged and involved. There is local prominence, but the symptom-complex of aneurism is not present. Caries, osteomyelitis, or actinomycosis may cause a rounded, fluctuating tumor near the sternum or ribs. In aneurism the bony structures of the chest wall may be recognized as overlying the tumor, and, when perforation has taken place, as entering into the formation of the opening through which the tumor protrudes, while in abscess they may be recognized as underlying the fluctuating tumor and separating it from the cavity of the thorax. In the very rare cases of mediastinal abscess the onset is abrupt, substernal

FIG. 364 — Bulging of the anterior wall of the chest in a case of mediastinal tumor. —Jefferson Hospital.

FIG. 365.—Abscess of the chest wall.—Jefferson Hospital.

pain is intense and persistent, and there are grave constitutional symptoms. *Pulsating Empyema.* — Intrathoracic pulsating empyemata give rise to more or less diffuse pulsation upon the left side in the anterolateral aspect of the chest. The signs of pleural effusion are present, the heart is usually displaced to the right, and Traube's semilunar space modified. Empyema necessitatis, when the tumor is in relation with the heart and pulsates, may simulate aneurism. The signs of left-sided pleural effusion, the fact that the tumor diminishes upon inspiration and increases upon expiration, the absence of heaving, forcible impulse, and diastolic shock, of the pressure symptoms incident to aneurism, and of tracheal tugging are conclusive. *Aortic Stenosis.*—A systolic murmur and thrill may suggest aneurism, but a consideration of the history of the case, the nature of the subjective symptoms, and the objective signs of the two conditions render the differential diagnosis an easy matter.

The Course.—The progress is irregular. Symptoms usually precede signs. There are periods of arrest. The contour of the sac and the pressure phenomena undergo from time to time changes due to changes in the accumulation of clot, its organization or failure to organize at different places, and uneven yielding of the wall under arterial pressure in various regions. In sacs that have perforated the chest wall such local expansions and retractions not rarely take place under the eye. They may be invoked in explanation of changes in the degree of dysphagia, the dyspnœa and stridor, the pupillary differences, and the locality and intensity of the pain. A knowledge of these facts justifies caution in ascribing diminution or even disappearance of certain pressure symptoms to an actual improvement in the underlying condition. Sacs that rapidly increase in size give rise to more urgent pressure symptoms and are more liable to early perforation than those whose growth is slow. The urgency of pressure symptoms depends in part upon the size of the sac and in part upon its location. Aneurisms of the ascending aorta cause compression of the trachea, a primary bronchus, or the œsophagus, only when they have attained considerable size; while a small sac in the concavity of the arch may compress the left bronchus, or a medium-sized sac springing from the inner aspect of the descending portion may cause difficulty in deglutition. Perforation occurs earlier in dissecting than in other forms of aneurism. In sacs containing much clot the perforation may be small and the bleeding moderate. The blood loss is often, however, rapid and fatal. It may be spontaneously arrested by a layer of clot which may permanently close the rent. As a rule, when hemorrhage is arrested it recurs from time to time and finally proves fatal. Rupture takes place most frequently into the pleural sac or a bronchus; less commonly externally, into the pericardium, the retroperitoneal connective tissue, or the peritoneal cavity; not often into the œsophagus, stomach, or intestine; and extremely rarely into the descending vena cava, the pulmonary artery, or an auricle. When the hemorrhage is free, death occurs at once with symptoms of internal bleeding. Perforation into the pericardium is usually fatal at once; into the retroperitoneal tissues it is usually delayed for a longer period. In the latter situation perforation is attended with intense pain and may at first simulate perforation peritonitis or embolism of the mesenteric artery. When the rent is small or the bleeding restrained by the adjacent structures, there are signs of internal hemorrhage, but life is prolonged for a period. I saw a patient who lived eight hours. Bleeding into a pleural sac causes the signs of an effusion; into a bronchus, hæmoptysis which may suggest phthisis or be moderate and prolonged, giving rise to a suspicion of pulmonary cancer; into the stomach, hæmatemesis; into the gut, bloody stools. Fragments of a thrombus may be detached, causing embolism, most commonly of a cerebral artery.

Prognosis.—Spontaneous cure cannot occur in fusiform aneurisms. Small sacs with narrow openings in rare instances become filled with clot which undergoes organization. In dissecting aneurisms a distant communication with the lumen of the aorta may be established, thus forming along the old course a new channel for the blood stream. These are rare events. In a majority of instances, aneurism of the aorta terminates in

death in the course of six months to three years. The average duration is about one year. Exceptionally life may be prolonged for several years. A time prognosis in individual cases is hazardous. Slowly developing and arrested aneurisms are of relatively favorable prognosis as compared with those of rapid growth, but unforeseen accidents may at any moment occur. Favorable conditions of life and treatment render, in exceptional instances, the prognosis less unfavorable.

FIG. 366.— Aneurism of the abdominal aorta.—*International Clinics.*

(b) ANEURISM OF THE ABDOMINAL AORTA.

Aneurism of the aorta is far less common below the diaphragm than above it. It may be fusiform or sacculated. In rare instances there are two or more —multiple aneurism. Still more rare in this region is dissecting aneurism. The most common situation is directly below the diaphragm and upon the anterior wall of the aorta, where the sac forms a distinct rounded tumor in the epigastric region in the median line and extending to the left. As the sac enlarges it projects into the left hypochondrium and may occupy a large part of the left side of the abdomen. When it projects posteriorly it causes erosion of the vertebral bodies, or may give rise to distinct spinal symptoms, and, increasing in dimension, may extend into the chest and rupture into the pleura or form an external tumor in the lumbar region. Perforation into the retroperitoneal space may give rise to a progressively enlarging hæmatoma extending into the left lumbar region and simulating a sarcoma.

Symptoms.—Pain is an early symptom. It is referred to the back and is often persistent, radiating to the left flank and marked by intense exacerbations, suggestive of intercostal neuralgia, or renal colic. In many of the cases there are pain-free intervals, or a dull, deep-seated pain in the back increased by movement or jarring of the body. The pain not rarely extends into the abdomen with exacerbations which simulate colic—belly-ache.

Physical Signs.—Upon INSPECTION epigastric pulsation may be present, frequently a diffuse prominence, and occasionally a distinct circumscribed tumor. PALPATION reveals a tumor mass, rounded, smooth, and the seat of forcible, expansile pulsation. A systolic thrill is common. PERCUSSION.—A large sac approaches the anterior wall of the abdomen and causes dulness, which may be continuous with that of the left lobe of the liver. AUSCULTATION reveals a systolic murmur usually best heard over the tumor and transmitted into the crural arteries, sometimes more distinct over the lower dorsal and upper lumbar vertebræ. In some cases there is also a diastolic murmur. These murmurs do not accompany the heart sounds, but follow them. Large aneurisms of the abdominal aorta displace the stomach and to some extent also the liver downward. Distinct retardation of the pulse in the crural arteries occurs when the sac is large.

Diagnosis.—DIRECT.—A distinct circumscribed epigastric tumor in the median line and extending to the left, which can be grasped and which is the seat of expansile pulsation, justifies a positive diagnosis of abdominal aneurism. In the absence of this symptom-complex the diagnosis cannot be made. It is of use to note that in aneurism the pulsation is epigastric, while dynamic pulsation is most marked immediately above or at the umbilicus.

DIFFERENTIAL.—*Dynamic Pulsation.*—The throbbing aorta is very common in nervous women. This pulsation is often very forcible and to a slight extent distinctly expansile, but it is not associated with a tumor, and the course of the aorta may often be felt upon palpation. A systolic murmur may be easily produced by the pressure of the stethoscope. Associated nervous symptoms, and the disappearance of pulsation under the influence of suggestion or other powerful psychic influences are of diagnostic importance. *Tumors of the Pylorus, Pancreas, or the Left Lobe of the Liver.*—New growths overlying the aorta in the epigastrium rise and sink with the movements of the arterial wall and are frequently mistaken for aneurism. The fact that the tumor is not expansile is of diagnostic importance. The absence of murmurs points to tumor rather than aneurism. The pressure of a tumor in contact with the aorta may cause stenosis and a systolic murmur. The crural pulse is not affected. The disappearance of pulsation in the knee-elbow posture, when the tumor falls away from the aorta, is an important sign. The X-rays are of less value as aids to diagnosis in abdominal than in thoracic aneurisms.

Prognosis.—Aneurisms of the abdominal aorta almost always end in death. In rare instances small sacs with a narrow communication with the lumen of the aorta have undergone obliteration by the deposit and organization of clots. Death commonly follows rupture, which may take place into the retroperitoneal tissues, pleura, peritoneum, or intestine. Far less frequently death is due to embolism of the superior mesenteric artery, complete occlusion of the aorta by clot, or paraplegia following erosion of the spine and compression of the cord.

ii. Aneurism of the Cœliac Axis and its Branches.

This branch is frequently involved in aneurism of the abdominal aorta. A negro, twenty-six years old, who was syphilitic, suffered from intense

paroxysmal pain radiating to the back and both sides of the abdomen. No tumor could be discovered. Death occurred suddenly with symptoms of internal hemorrhage. At the autopsy there was found an aneurism of the axis the size of a small orange, which had ruptured into the peritoneum. The splenic artery is occasionally the seat of small, sometimes of large, aneurismal sacs. Epigastric pain, vomiting, hæmatemesis, and hemorrhage from the bowel may occur. A deep-seated tumor extending to the left with or without pulsation, and dulness reaching to the spleen, are significant. Rupture into the colon may occur. Aneurism of the superior mesenteric artery may cause a movable pulsating tumor, which is often the seat of a systolic murmur but is not associated with retardation of the crural pulse. Symptoms of mesenteric infarction may occur. Aneurism of the hepatic artery is extremely rare. The symptoms are obscure and a positive diagnosis is impossible. Rupture into the bile passages may occur. The differential diagnosis between aneurisms of these arteries and aneurisms of the aorta is commonly attended with insurmountable difficulties.

iii. Arteriovenous Aneurism.

That form which results from the rupture of an aneurism of the ascending portion of the arch of the aorta into the descending vena cava is of special clinical interest. It is, however, extremely rare. The symptoms usually occur abruptly and consist of marked dilatation of the veins of the upper part of the body, with cyanosis and œdema. A continuous murmur with systolic intensification and a systolic thrill may be recognized.

iv. Periarteritis Nodosa; Congenital Aneurism.

The medium-sized arteries, especially those of the muscles, and the heart, spleen, liver, kidneys, intestines, and the skin are the seat of whitish nodular masses, varying in size from a small shot to a large pea, and great numbers of small aneurismal dilatations. The disease has occurred in both sexes. It manifests itself most commonly in early and middle adult life. It has been ascribed to syphilis and to septic conditions. It is extremely rare. The prominent symptoms are weakness, anæmia, and rapidity of the pulse. There is at first fever, which presently falls without a corresponding decline in the pulse-frequency. When the muscular branches are involved, pain, weakness, and atrophy occur. When the arteries of the gastro-intestinal canal are the seat of the lesions, epigastric pain, thirst, anorexia, nausea and vomiting, and diarrhœa or constipation are prominent symptoms. Hemorrhage from the bowels has been observed. Scanty urine, of low specific gravity, albumin, and casts occur. Urea is diminished. Anæmia is marked. Leucocytosis is common. The course of the disease is progressive, and death occurs at the end of the second or third month. Recovery is exceptional. The diagnosis is usually post mortem. The nature of the affection may be suspected when, in connection with the foregoing symptom-complex, nodular thickenings may be felt in the course of accessible arteries.

XIV.

THE DIAGNOSIS OF DISEASES OF THE NERVOUS SYSTEM.[1]

DISEASES OF THE BRAIN.

I. MENINGITIS.

Inflammation of the membranes of the brain is common to a variety of affections. Thus, there are purulent meningitis, tuberculous meningitis, syphilitic meningitis, and meningitis due to various other microbian infections, or associated with disease of bone. For descriptive purposes we distinguish between inflammation of the dura—*pachymeningitis*—and inflammation of the pia,—*leptomeningitis,*—although the two membranes are often involved together.

Inflammation of the dura mater may occur from septic infection, and may be purulent. The commonest causes are injury, infection, and necrosis of bone. Thus disease of the middle ear is the most frequent cause, but cases may arise from infection through the nose by way of the cribriform plate. Fracture of the skull may sometimes be the starting-point. Large quantities of pus may be present, and the pia mater and brain substance may be involved as well as the dura. There is a variety in which the pus is localized or pocketed between the skull and the dura, usually secondary to injury of the skull or to caries or bone syphilis—the so-called *pachymeningitis externa.*

A distinct form of pachymeningitis is the disease known as hæmatoma of the dura mater—the *pachymeningitis hæmorrhagica* of Virchow. It occurs especially in the chronic insane and in old alcoholic patients. There is observed beneath the dura a layer, quite thick, which looks like organized blood-clot; this often exists, in fact, in several layers, as though caused by successive hemorrhages. Sometimes the appearance is that of a very delicate vascular membrane, enclosing in its meshes blood which is more or less organized. Cysts may be found and other evidences of breaking down of the new tissue.

The **symptoms** of pachymeningitis hæmorrhagica are obscure, being masked by the general condition of the patient, who is usually a chronic lunatic or a confirmed inebriate. A similar lesion has been described after sunstroke, and may account for the headache and mental changes.

Simple, idiopathic, or isolated inflammation of the pia mater is such a debatable condition as scarcely to be considered a clinical entity. The forms of leptomeningitis which we usually distinguish at the bedside are those that occur in septic infection, such as in otitis media, and in cerebrospinal fever, syphilis, and tuberculosis. A very characteristic form is that seen in general paresis, in which the pia mater is thickened and opaque and so adherent to the summits of the convolutions that it is stripped with difficulty, and usually carries away with it a portion of the brain substance. Quincke has described a *serus meningitis* in which the pia-arachnoid

[1] Contributed by Dr. Lloyd as collaborator.

especially is involved; it is characterized by mild symptoms, slight fever, and headache, with some stiffness of the back of the neck; and optic neuritis is not uncommon with it. Meningitis has been observed in mumps, and it may also be caused by the pneumococcus.

Symptoms.—The general symptoms of leptomeningitis are headache, fever, delirium, convulsions, rigidity of the neck and face muscles, exaggerated reflexes, followed later by paralysis and coma.

Tuberculous Meningitis (see page 186).

Epidemic Cerebrospinal Meningitis (see page 126).

Septic meningitis may be caused by otitis media, in which case the microbe is usually a streptococcus or staphylococcus; or it may be caused by the pneumococcus. This meningitis is associated with pus formation, also with pachymeningitis and purulent cerebritis, and in not a few cases leads to brain abscess.

Diagnosis.—Meningitis, whether in the dura or the pia mater, depends upon so many causes and is associated with so many pathological processes that it is difficult to lay down uniform rules for the diagnosis. The first essential is to seek for the cause, such as a middle-ear disease, tuberculosis, meningococcic or other infection, injury to the bones of the skull, caries, sunstroke, syphilis, etc. The condition described below as acute hemorrhagic encephalitis, due to some infection, may be present.

Symptoms of irritation appear early. These are headache simulating neuralgia, vomiting, optic neuritis, fever, stiffness of muscles, and perhaps convulsions, with irritability of temper, change of disposition, and even somnolence, delirium, or stupor. Later the symptoms are indicative of pressure and profound toxæmia: thus we see various paralyses, increasing stupor merging into coma, rapid pulse, and failing powers.

Meningitis of various kinds may be mistaken for mere neuralgia, but the history and associated symptoms of organic disease should prevent error. Where headache, vomiting, and constipation are early symptoms, as in tuberculous meningitis, the case may suggest gastric or intestinal disorder, but the onset of more pronounced brain symptoms, such as convulsions, stupor, and various paralyses, is unmistakable. The best single general rule for the distinguishing of meningitis is to be on the lookout in suspected cases for the signs of infectious diseases of the brain, and to trace these to their cause. Lumbar puncture may shed important light in cases of infection, and may show an opaque or cloudy cerebrospinal fluid, globulin, lymphocytosis, and even polymorphonuclear cells and organisms, according to the nature of the case. These findings are described more in detail elsewhere.

II. ACUTE HEMORRHAGIC ENCEPHALITIS.

The disease is characterized by multiple foci of congestion and both punctate and massive hemorrhages, with infiltration of leucocytes, minute emboli, and localized necrosis of the brain-tissue. The membranes are usually not involved. The reported cases have mostly followed the infectious diseases, such as influenza, mumps, erysipelas, pneumonia, typhoid and typhus fever, and malaria. Nephritis has been observed in some cases. The disease is probably caused by the local action of micro-organisms or by their toxins. Oppenheim called attention to the resemblance of the disease process to the polioencephalitis superior of Wernicke and to the

polioencephalitis anterior of Strümpell. The possible association with poliomyelitis should not be ignored.

Symptoms.—The symptoms are suggestive of meningitis, which, however, is not always present. There are headache, vomiting, convulsions, localized palsies, and affections of consciousness. Rigidity of the neck sometimes occurs. Fever is not constant, and there may even be subnormal temperature. The pulse at first may be slow, but in fatal cases the pulse becomes rapid, the respirations shallow, coma sets in, and death occurs in a few days or weeks. Recovery has been claimed in a few cases.

Diagnosis.—The disease is likely to be confounded with meningitis, and the distinction between the two is not of practical importance, as the course and treatment are nearly the same. In most cases an accurate diagnosis has only been made after death. This form of encephalitis may be suspected when grave cerebral symptoms occur in the course of, or immediately following, any of the infectious diseases mentioned.

III. PURULENT MENINGO-ENCEPHALITIS AND BRAIN ABSCESS.

Pus-forming inflammation within the cranium assumes several forms. There may be a diffuse purulent meningitis, or, as is more common, a meningo-encephalitis; or there may be abscess. The clinical distinction between these forms is not easy.

Etiology.—The causes are the various pyogenic micro-organisms. One of the commonest is seen in purulent otitis media; and more rarely other cranial bones, especially the bones of the nose and the orbit, and their sinuses, may be the starting-point. Accessory sinuses of the nose are frequently infected, and this infection may travel by way of the frontal sinus, sphenoidal sinus, ethmoid cells, or the antrum of Highmore. The streptococcus is the chief germ in otitic brain abscess. Septic infection, arising from any focus in the body, may cause a metastatic brain abscess, but this complication is rather more common in ulcerative endocarditis, abscess of the liver, abscess and gangrene of the lung, and empyema. Trauma of the skull may also cause abscess of the brain. Suppurative meningitis occurs in tuberculosis and in cerebrospinal fever, and it may also be caused by the pneumococcus.

Pathology.—The membranes, especially the pia-arachnoid, are inflamed and opaque in purulent meningitis, and pus is diffused beneath them. This pus often follows along the perivascular spaces and the fissures of the brain, appearing as white streaks. In some cases it is more copious, and forms collections beneath the membranes, especially at the base. The cerebral tissue is often involved, the gray and white matter being infiltrated, œdematous, and softened in places. There may also be sinus thrombosis, with engorgement of veins on the outside surface of the skull. When abscess forms it is either diffuse or circumscribed. In the former case the collection of pus is merely an accident of a more or less diffused purulent meningitis. The circumscribed abscess is contained within a limited area, and its walls may even be thickened, forming a barrier between the pus and the brain tissue. These circumscribed abscesses are most common in the temporal lobe of the cerebrum and in the cerebellum. In some cases multiple abscesses are formed, especially in metastasis.

Symptoms.—These are general and focal. Among the general symptoms we include those which indicate a grave cerebral disorder; and among the focal symptoms those which indicate its location in the brain. The *general* symptoms are fever, headache, vomiting, convulsions, paralysis, optic neuritis, and affections of consciousness. Fever is not constant nor always of one type; it may be slight and easily overlooked; again it may be more pronounced, and accompanied with chills and sweating. It may depend as much on the primary condition (otitis media, abscess of the lung, etc.) as on the brain lesion. *Per contra*, in some cases of brain abscess the temperature is subnormal and the pulse very slow. Headache is usually present in suppurative meningitis, but it is not an altogether constant symptom in brain abscess. It was present in 78 out of 169 cases in Allport's table. In some cases, however, it is an early indication of an insidious onset. Its localizing value is not always great; in cerebellar abscess, for instance, the pain has been observed in other parts of the cranium, even in the frontal region. Occasionally, however, the pain is strictly localized at the region of the abscess, and pressure and percussion on the skull at that point may be painful. Vomiting is frequent in cerebellar abscess, but more rare in diffuse purulent meningitis; and it is usually of the cerebral type—propulsive and not dependent on food in the stomach. Convulsions may or may not be present; there is no positive rule about them. They sometimes have localizing value. The same can be said of paralysis. Optic neuritis is a most important symptom of abscess of the brain, although not present in all cases. In children, if the pus formation be rapid, optic neuritis will soon ensue. It has no localizing value, but its presence in cases of latent or suspected abscess is most significant. Affections of consciousness are common in all forms of suppurative disease of the brain, and they range from mere apathy and stupor to wild delirium and profound coma. In cases of latent abscess changes in the temper and personality, such as irritability and depression, are observed.

By the *focal* symptoms we attempt to determine the site of the abscess. These symptoms are mostly pain, paralysis, and convulsions.

Pain, as already said, is not altogether reliable as a guide. Both in cerebellar and temporosphenoidal abscess the pain is sometimes frontal, or it may be more generally diffused. When sharply localized, as in the occiput or nuchal region, especially if it is increased by percussion, it may prove a safe guide.

Paralysis may be clearly indicative of the site, especially if the abscess involves the motor regions, as the pre-Rolandic area and its subcortical connections. This may occur either in frontal or parietal abscess, the pus extending backward or forward respectively. We then have hemiplegia or monoplegia (facial, brachial, or crural), according to the centres involved, and in left-sided lesions there is aphasia. Sensory aphasia, especially word deafness, is strongly indicative of a temporal abscess on the left side, such as is common to ear disease, and it has led the way to successful operation. Temporal abscess may also give rise to hemiplegia by pressure across the Sylvian fossa upon the internal capsule; hence this symptom may be misleading. Hemiplegia has even been caused by abscess of the

cerebellum. The hemiplegia in such cases is probably caused by pressure on the pons or medulla oblongata. In this way also cerebellar abscess may cause other pontile symptoms, as paralysis of the fifth, sixth, seventh, and eighth nerves, and the mid-brain may even suffer, with a consequent partial ophthalmoplegia. According to Allport's table strabismus occurred in 10 out of 98 cases of abscess of the brain, not all cerebellar. From these various data it is seen that paralytic symptoms must be interpreted with care. For instance, thrombus of the cavernous sinus, which might occur in any purulent process in the brain, causes an ophthalmoplegia, as may also an abscess of the temporal lobe by pressure on the third and sixth nerves.

Involvement of the sensory tract, causing hemianæsthesia and hemianopsia, has been reported, especially in abscess of the temporal lobe. Various affections of the visual fields may be caused by abscess in the occipital lobe.

Focal epilepsy usually indicates a lesion in or about the motor region, but this symptom is not so common as in brain tumor. Unilateral convulsions have been reported in cases of abscesses at various sites, even in the cerebellum. Tonic spasm in the nape of the neck may be caused by abscess of the cerebellum.

When a thrombus forms in a sinus the veins on the outside of the skull may be congested: thus in thrombus of the cavernous sinus the veins of the orbit are engorged; and the veins at the base of the nose and on the brow are sometimes congested from thrombus of the superior longitudinal sinus.

Among other noteworthy symptoms are vertigo, incoördination, loss of equilibration, especially but not always in cerebellar abscess, and abolished knee-jerks. Macewen has noted this last symptom in cases of cerebellar abscess, and it has been seen in cerebellar tumor, especially of the middle lobe. Occasionally it is not continuous, the reflex disappearing and reappearing. Knapp has seen a case of cerebellar lesion in which only one knee-jerk was lost, and that on the side opposite to the lesion. Abscess of the frontal lobe may cause obscure mental symptoms, especially retarded cerebration. In Allport's collection of 98 cases of abscess of the brain, 40 occurred in the temporal lobe, 31 in the cerebellum, 7 in the parietal lobe, and the remainder in various other regions. In 5 cases there was diffuse subdural abscess.

Diagnosis.—Purulent meningitis and brain abscess, although included here under one heading and having much in common, should be distinguished from each other if possible. The two conditions may coexist, or the one merge into the other, and the dividing line is not easily detected. Focal symptoms are not likely to be seen in diffuse purulent meningitis, and the course of this disease is more acute and rapid; in fact, death may occur in a few days. Abscess is likely to be much slower, especially the circumscribed abscess with well-formed walls; and in this connection the latent abscess must not be overlooked; this may endure for weeks, even months, with only very obscure symptoms. In fact, a latent or premonitory stage is not uncommon in solitary abscess of the brain, but in diffuse abscess, and especially multiple abscess, such as is

caused by metastasis from some septic process outside the brain, the case is likely to be more rapid and more like a diffuse suppurative meningo-encephalitis.

Some authors attempt to distinguish extradural abscess, such as may occur in trauma and even in otitis. The local symptoms of such an infection are usually evident, as pain, swelling, venous engorgement. The presence of an external wound is important.

Tumor of the brain is distinguished from abscess by its different clinical history, its slower onset, the absence of a septic process of origin, and, as a rule, of chill and fever. But in rare cases tumor of the brain is complicated with suppuration. Optic neuritis is rather more common in tumor, but only little reliance can be placed on that fact. The evolution of symptoms is usually more gradual and progressive in the case of tumor.

Cerebellar abscess may simulate Ménière's disease by vertigo and occasional deafness. The clinical history, however, is different, and in case of abscess there are likely to be fever, headache, and mental changes.

Cerebral hemorrhage and softening may simulate abscess when the latter is fully formed, but the clinical history is so entirely different that the distinction is easy, as a rule. An abrupt onset of symptoms may occur in case of latent abscess, if the pus breaks from its cavity, causing paralysis, convulsion, coma, etc.; and in such a case the differentiation is perplexing.

In all cases of septic infection, such as suppurating otitis, abscess of the lung, etc., the onset of cerebral symptoms should excite suspicion, and the case should be carefully scrutinized. Lumbar puncture may give valuable information, as described elsewhere (page 131).

IV. SINUS THROMBOSIS.

Thrombosis of the cranial sinuses is either primary or secondary. In the former the thrombus arises from some general blood state, in the latter from some disease, usually septic, in the immediate neighborhood.

Etiology.—Primary thrombosis is seen in conditions of exhaustion, often as a terminal symptom; thus it occurs in advanced stages of tuberculosis, carcinoma, the infectious diseases, as typhoid fever, and in the diarrhœas of infancy. It is favored by a weakened heart and by the sluggish circulation in the sinuses. It has been seen also in chlorosis and anæmia.

Secondary thrombosis results from disease of the walls of the sinus, hence especially from injury or caries of bone. Its commonest cause is otitis media, in which case the lateral and transverse sinuses especially are involved. It also arises by way of the nasal bones, and in fact, though rarely, from any other bones of the cranium which become the seat of caries. It is sometimes caused by fractures; and it also arises from septic processes within the skull, such as a purulent meningitis from any cause, or from a general septicæmia or pyæmia arising from causes outside the cranium. Facial erysipelas may be a cause.

Pathology.—In septic or secondary thrombosis the sinus is partly or entirely filled with a white or grayish-white mass, adherent to the walls.

This mass may be purulent and sanious, and the walls of the sinus are inflamed, infiltrated, and discolored, while the contiguous bone in many cases, as in otitis, is carious and softened. In recent cases the thrombic mass is soft and easily broken up, but in older cases it is quite firm and fibrous. It may extend for some distance through the sinus, even into some of the tributary veins, thus causing engorgement of veins, with swelling and œdema, on the outside of the skull. These septic thrombi are not seldom associated with other septic lesions, such as purulent meningitis, purulent encephalitis, and even brain abscess.

Symptoms.—These are general and local. In the case of the secondary septic thrombi the *general* symptoms may be masked by those of the general pyæmia; thus there is fever, usually of a septic type, with headache and changes in consciousness, and there are not seldom the evidences of meningitis. Convulsions sometimes occur, and various paralyses, and occasionally a high grade of choked disk. In the case of primary thrombosis we have to consider the original disease and its exhausting effects, but superadded to these we observe grave cerebral symptoms of sudden onset, such as headache, vomiting, stupor, followed by a gradually deepening coma, possibly with convulsions; but local paralytic symptoms are rare in this form. There may, however, be hemiplegia, or even a general flaccid paresis.

The *local* symptoms are sometimes conspicuous. They depend usually upon engorgement of tributary veins on the outside of the skull and upon paralysis of certain cranial nerves.

In thrombosis of the cavernous sinus there are protrusion of the eye, swelling and discoloration of the tissues about the eye, engorgement of the veins of the orbit and the frontal veins, which communicate through the orbit with this sinus, possibly choked disk, and paralysis of the third, fourth, and sixth nerves, which run through the sinus. There may also be pain or anæsthesia in the ophthalmic division of the fifth nerve. The central retinal vein may also be the seat of a thrombus.

Thrombosis of the superior longitudinal sinus may cause cyanotic swelling and œdema on the brow, and in rare cases nose-bleed. Infection of this sinus may occur through the nasal bones and be accompanied with a purulent meningo-encephalitis. The commonest site of cranial thrombosis is in the lateral and transverse sinuses in cases of otitis media. The local symptoms are disguised by the local bone disease. The most significant are pain and œdema over the mastoid. Otitis media is the most common cause of grave general infection of the cranial contents, the most serious being abscess. Irritation from this focus may cause recurring epileptic fits. The jugular vein, external or internal, may be the seat of thrombus, which may even be palpable. A gravity abscess may simulate phlebitis of the jugular; and the glossopharyngeal, vagus, accessory, and hypoglossal nerves have been paralyzed in some of these cases.

Diagnosis.—A local diagnosis is hardly practicable for any other of the cranial sinuses than those mentioned above. It is, of course, much simplified in cases in which circumscribed œdema occurs. The general diagnosis is often difficult and problematical. Thrombosis is to be suspected when grave cerebral symptoms, such as headache, vomiting, con-

vulsions, stupor, and unconsciousness, occur suddenly in cases of septic infection, such as otitis media and facial erysipelas, and in wasting diseases, such as tuberculosis, carcinoma, infectious diseases, and infantile diarrhœa. But a differential diagnosis from meningitis, abscess, and softening is not always possible, and in fact some of these conditions may be associated with thrombosis. Thrombi of the cerebral veins are sometimes the cause of hemiplegia and diplegia in young children. Smithers has recently called attention to hemiplegia in typhoid fever, caused by thrombi in the cerebral arteries.[1]

V. CEREBRAL HEMORRHAGE.

Etiology.—The cause of this accident is primarily some disease of the blood-vessels, excluding, as we do here, hemorrhage from trauma. The diseases of the blood-vessels are chiefly arterial sclerosis or atheroma, occurring usually after middle life, and more rarely syphilis, occurring at any period of life and not rarely in young adults. It is not to be overlooked, however, that the hemiplegia of syphilis is usually due to an inflammation and thickening of the walls of a blood-vessel rather than to a hemorrhage.

Pathology.—Atheroma of the blood-vessels is a common affection in later life. It is rare to see an autopsy in a person past fifty without some evidences of it, and in persons of sixty and seventy it is not unusual to see the circle of Willis at the base of the brain so thickened and hardened that the vessels are like pipe-stems. From these main arteries at the base the branches that pass up through the anterior perforated space to the lenticular nucleus and internal capsule are especially liable to suffer. More rarely the branches from the posterior cerebral or those from the basilar, vertebral, and cerebellar arteries are affected. The atheromatous arteries are frequently the seat of minute dilatations, aneurismal in character, and it is one of these that is likely to give way. The hemorrhage is usually within the substance of the brain; meningeal hemorrhage from arterial disease being rare, although common from trauma. Occasionally, however, a hemorrhage breaks through to the surface. The most common seat of hemorrhage is in the lenticular nucleus, which is a part of the basal ganglion (corpus striatum) within the brain. The hemorrhage occurs in such a way as to press upon or destroy the internal capsule which contains the motor and sensory tracts. The weakened artery at this point was called by Charcot the "artery of cerebral hemorrhage."

In recent cases the blood is either still fluid or partly clotted, and it occupies a ragged cavity which it has torn out of the substance of the brain. In old cases this cavity is often found walled off, forming a cyst, filled with a reddish or yellowish fluid. In rapidly fatal cases it is sometimes found that the blood has broken through into the lateral ventricle, or even to the outer surface of the brain. Multiple hemorrhages may occur, and sometimes the hemorrhage is in the parietal or occipital lobe, or even in the pons. Hemorrhage in the cerebellum is less common than in the cerebrum.

[1] Journal of the Am. Med. Assn., Aug. 3, 1907, p. 389.

Instead of hemorrhage a diseased blood-vessel may cause thrombosis. There is then secondary softening, but clinically the two conditions are much alike and it is quite impossible, as a rule, to distinguish them. Softening is also caused by embolism, with very similar results.

In long-standing cases of hemiplegia there occurs a descending degeneration of the motor tract, which may be traced through the peduncle, the pons, the decussation in the medulla, and the spinal cord.

FIG. 367.—Old left hemiplegia with contractures. —Lloyd.

Symptoms.—Cerebral hemorrhage causes what is popularly known either as a "stroke" or an "apoplexy." These two conditions are distinguished chiefly by the state of the consciousness; in the former the mind may be clear, in the latter there is stupor or coma. In either case there is likely to be paralysis, according to the site of the lesion.

In the lenticular nucleus and internal capsule, the most common site, hemorrhage causes hemiplegia on the opposite side. The arm and leg are paralyzed, the arm rather more so, and in some cases the lower part of the face and one side of the tongue. The upper portion of the face is not involved, so the patient can still shut his eyes and wrinkle his forehead. The tongue, if involved, is protruded toward the paralyzed side. In some cases there is hemianæsthesia, and even hemianopsia, the affection of the sensory fibres showing that the clot has involved the posterior portions of the internal capsule. There may also be various forms of aphasia if the lesion is in the left hemisphere.

When the hemorrhage involves the island of Reil and the posterior end of the third frontal convolution on the left side there is motor aphasia. When the left superior temporal convolution is involved there is word-deafness and verbal amnesia: and when the left angular gyrus is invaded there is word-blindness and object-blindness. Various mixed forms of aphasia, the so-called sensorimotor aphasia, may occur from hemorrhage into various portions of these speech centres (the so-called language zone) and their subcortical connections in the left hemisphere.

Hemorrhage in the frontal lobe, if it does not involve the motor centres or tracts, may cause very obscure symptoms, more especially mental changes, such as retardation of the mental processes, loss of the power of attention, etc.

Hemorrhage in the superior parietal lobule may cause ataxia of the limbs on the opposite side, and sensory changes, especially astereognosis.

Hemorrhage in the occipital lobe may cause hemianopsia and other partial defects in the visual fields, and also some inability to recognize and name objects by sight.

In the cerebellum hemorrhage may cause intense vertigo, loss of equilibration, forced and pitching movements, and vomiting; and if the clot is big enough to make pressure on the mid-brain and pons, there may be hemiplegia, hemianæsthesia, and involvement of the oculomotor, fifth, sixth, seventh, and eighth nerves; but these latter symptoms are rare, and are rather indicative of either a mid-brain or a pontile lesion. Thus hemorrhage in the pons causes the hemiplegia alternans, in which the arm and leg are paralyzed on the opposite side while the sixth, seventh, eighth, and possibly the fifth nerves are paralyzed on the side of the lesion; this is so especially if the lesion is low in the pons. If the lesion is high, the cranial nerves named may be paralyzed on the opposite side, that is, on the same side as the hemiplegia. In rare cases a very circumscribed lesion that involves the nucleus of the sixth nerve may also cause diabetes or polyuria.

In hemorrhage in the mid-brain (corpora quadrigemina and cerebral peduncles) the oculomotor and fourth nerves may be paralyzed on the side of the lesion, while the hemiplegia is on the opposite side, presenting a type of hemiplegia alternans which is sometimes called the "syndrome of Weber."

Hemorrhage in the medulla oblongata is extremely rare, and is quickly fatal if the respiratory centres are involved.

In the apoplectic state consciousness may be partially or entirely lost; the breathing becomes stertorous, the cheeks puff out with every breath, and the pulse may be full and strong. If the case advances toward an unfavorable ending, the pulse becomes thin and rapid, the temperature rises, unconsciousness is profound, the pupils do not react to light and may be unequal. Cheyne-Stokes respiration may set in, and death is often hastened by an œdema of the lungs. In the apoplectic cases the hemiplegia can sometimes be determined by the loss of resistance to passive motion on the paralyzed side.

In some cases the reflexes are not at first greatly affected. In cases of massive hemorrhage with shock and unconsciousness, the knee-jerk on the paralyzed side may be abolished. In patients who survive and partially recover, the deep reflexes become exaggerated on the paralyzed side, the muscles are contractured, and there results a characteristic hemiplegic attitude and gait. Ankle clonus and Babinski's reflex are usually present in these patients.

The state of the pupils is not constant; in the early stages the light reflex may be preserved; but with deep unconsciousness it is usually abolished. The pupils are sometimes slightly unequal; or they may be of normal size or even dilated. In pontile hemorrhage they may be contracted.

Lateral deviation of the head and eyes is sometimes seen in the apoplectic cases, the head and eyes being turned away from the paralyzed side—conjugate deviation. If a spastic state sets in from irritation of the brain-cortex or motor tracts, especially if convulsions occur, as sometimes happens, the head and eyes are forcibly drawn towards the paralyzed side. In rare cases there results a "posthemiplegic chorea"—a bad term, as the disorder is not a true chorea, but a wide to-and-fro tremor.

Diagnosis.—Cerebral hemorrhage, especially when it causes unconsciousness, requires to be distinguished from uræmia, diabetic coma, postepileptic coma, opium poisoning, alcoholic drunkenness, and trauma. The problem is sometimes a difficult one. As a general rule hemorrhage causes a hemiplegia, which can usually be determined, even in cases of unconsciousness, by some difference in the resistance to passive motion on the two sides: on the paralyzed side the limbs are entirely flaccid and fall dead, while on the other side there is usually some resistance. In profound unconsciousness, however, the difference may be difficult to recognize. In cases of a simple paralytic "stroke" without unconsciousness the problem is much simplified, as the history of a sudden attack of hemiplegia is usually determinative. Even in these cases there may be at first some confusion of mind and clouding of consciousness.

Uræmic coma is often ascertainable from the history of the case. The presence of albumin and casts in the urine cannot determine the question positively because a patient with nephritis may have a cerebral hemorrhage or thrombus, while a patient with apoplexy may have albuminuria. Moreover, there are sometimes seen in nephritis attacks of hemiplegia, which pass away with other uræmic symptoms. Transient aphasia and brachial monoplegia of uræmic origin sometimes occur and may simulate organic lesion in the brain. In uræmic coma there may be prolonged subnormal temperature. In spite of the exceptions noted, uræmic coma is, on the whole, indicated by the state of the urine, the history of the case, the subnormal temperature, the usual absence of hemiplegia and other symptoms of a focal lesion, and sometimes by the uræmic odor. Convulsions are in favor of uræmia, although they sometimes occur in hemorrhage. Albuminuric retinitis is also in favor of uræmia. There are doubtful cases which only time can solve.

Diabetic coma is indicated by the glycosuria, diaceturia, and acetonuria, the history of the case, and the absence of hemiplegia. The crisis may be ushered in with headache and delirium, and the peculiar dyspnœa, called by Küssmaul "air-hunger," but these prodromes are not seen in every case.

Postepileptic coma usually clears up in a few hours at most. The history of the case is significant. Convulsions may occur in cerebral hemorrhage, but they are rare. Focal epilepsy, in which a hemiplegia or monoplegia may persist for some time, may be puzzling, but the history of the case and the course should prevent error, especially if focal symptoms are caused by organic lesion, such as tumor. In epileptic "status" the fits recur at frequent intervals (as many as twenty-five and even more in a day), the patient being profoundly unconscious between the paroxysms, with weak pulse, and sometimes with high temperature.

Opium poisoning is, as a rule, easily recognized by the history, the contracted pupils, and the slow respiration; in cases in which the history is unknown mistakes are possible. Massive hemorrhage, especially ventricular hemorrhage, may cause profound unconsciousness, immobile rather than contracted pupils, and labored breathing, while the hemiplegia may be masked; pontile hemorrhage is said particularly to simulate opium poisoning, especially in the contracted pupils. The extremely slow breathing of opium narcosis, however, is not likely to be seen in hemorrhage; while in the poisoning there is never hemiplegia, nor conjugate deviation of the head and eyes; and, finally, the extreme bilateral myosis is hardly equalled in cases of apoplexy, in which inequality of the pupils is more common. The pupil dilates as death approaches in opium poisoning.

Alcoholic intoxication is known by the history, by the odor of alcohol, and by the fact that the stupor or unconsciousness is usually not so profound as in apoplexy. None of these data is entirely reliable. A drunken man may have a cerebral hemorrhage, hence the odor of alcohol on the breath is a most unreliable test for this, as for any case. Hemiplegia is, of course, conclusive as against mere alcoholism, and unequal pupils suggest apoplexy. A few hours usually determine whether a doubtful case is one of drunkenness.

Trauma may cause a condition closely simulating or even identical with apoplexy, as, for instance, when it causes a cerebral hemorrhage. The history is most important. In all cases a careful inspection of the scalp and skull should be made in order to detect contusion or fracture.

Hysteria may possibly simulate apoplexy, but only superficially. The unconsciousness is usually not profound; the patient is often open to suggestion; there may be characteristic stigmata; the pupils respond freely to light; and deep ovarian pressure usually brings some response. Iridoplegia, however, has been observed in hysteria, but it is a very rare symptom and difficult to explain.

The differential diagnosis between cerebral hemorrhage and embolism is difficult and may be impossible. At most there are suggestions, not positive grounds, for an opinion. Hemorrhage usually occurs in persons at and beyond middle life; embolism in persons at any age in whom there has been a vegetative endocarditis. Hemorrhage is rather more prone to cause loss of consciousness than is an embolus. Associated cardiac and renal disease, being productive of diseased blood-vessels, may cause hemorrhage. The same is true of syphilis, although syphilitic hemiplegia is more frequently caused by meningitis and endarteritis at the base of the brain.

The Basal Ganglia.

These are the corpus striatum and optic thalamus, lying within the cerebral hemisphere. The former is divided by the internal capsule, containing the motor and sensory tracts, into the lenticular and caudate nucleus. As already explained, the blood supply is from the vessels coming up through the anterior perforated space from the large arteries at the base of the brain. Kinnier Wilson has described a "progressive lenticular degenera-

tion," a familial affection, associated with cirrhosis of the liver. The symptoms are very similar to those of pseudobulbar palsy, which is also caused by lesions here (*q. v.*). There is dysarthria, dysphagia, tremor, muscular rigidity and automatic laughter or crying. It is claimed that the pyramidal or motor tracts in the capsule are not involved; but this may be doubted, for even if not grossly impaired, they may nevertheless be interfered with by the vascular lesions in the lenticula. A "steadying" or "tonectic" influence is ascribed to the striatum, or to systems of fibres within it, by Wilson, Mills, and others. There is practically nothing known about the functions of the corpus striatum, and Lloyd claims that this body is vestigial, and merely represents the original fore-brain mass in the earliest ancestral types of the vertebrates. Lesions within its substance only act by interfering with the neighboring internal capsule.[1]

A thalamic syndrome has been described for lesions of the thalamus. It consists of hemianæsthesia, with pain, on the opposite side, with certain disorders of motion, such as ataxia or athetosis, with hemiparesis. These symptoms vary considerably in different cases. The claim that the thalamus is a centre for automatic movements of expression (laughter and weeping) is not proved, and is most doubtful.

VI. CEREBRAL SOFTENING.

Softening may result from any process that obstructs a blood-vessel; hence an endarteritis, causing thickening of an artery, as in syphilis; a thrombus from disease of the arterial walls, as in atheroma; and finally, an embolus, from the vegetations of endocarditis—all these may cause cerebral softening.

Pathology.—Thrombus and embolus produce results so nearly identical that they cannot be distinguished clinically. The special importance of embolus, from the clinical standpoint, lies in the fact that it may occur in young persons and produce all the symptoms of an apoplectic or hemiplegic stroke. It occurs in vegetative endocarditis. It is also common in malignant endocarditis, and sometimes occurs in other forms of sepsis. Thus hemiplegia may happen in the puerperium. Thrombosis occurs in some blood states other than sepsis, such as anæmia and chlorosis; it is most common, however, in diseases of the coats of the blood-vessels, as atheroma and syphilis. The vessels most involved are those that form the circle of Willis or some of their branches, especially the middle cerebral artery. Softening of the cerebellum and pons is occasionally seen.

The area involved does not always break down at once; in fact a comparatively long time may elapse before it softens. This leads to deceptive appearances at autopsies. When the tissue has once become soft it may be quite diffluent, and in color may be white, yellow, or red, according to the amount of blood elements contained in it.

Symptoms.—These are similar to those caused by hemorrhage. Hemiplegia is the commonest result, but other paralyses, such as monoplegia, hemianæsthesia, and hemianopsia, occur, as in hemorrhage, according to the seat of the lesion. Aphasia is not uncommon when the softening occurs in

[1] The Morphology and Functions of the Corpus Striatum. Jour. Nerv. and Ment. Dis. June, 1915.

the lenticular nucleus or the cortical speech centres or in their subcortical connecting tracts in the left hemisphere. Apoplectic symptoms, with confusion, stupor, or loss of consciousness, may be present if a large area is involved, but they are rather more uncommon than in hemorrhage. Occasionally the onset of symptoms is gradual and the course progressive, especially if small successive thrombi occur, but usually in the case of embolus the onset is sudden. Prodromes also occur in case of atheroma, such as vertigo, headache, and failure of memory and other mental powers. In some cases we see transient hemiplegia and other paralyses, due doubtless to the fact that the circulation, after being obstructed, may be restored. But in most cases there is left some permanent loss, such as hemiplegia and aphasia. Thrombus of the carotid or of the basilar artery causes grave symptoms, such as profound unconsciousness and failure of respiration; and in the case of the basilar artery there may at first be staggering, ataxia, or even a cerebellar gait.

Fig. 368.—Softening of the brain in the motor area of the right hemisphere, due to embolus.—Lloyd.

Diagnosis.—The distinction between hemorrhage and embolus largely depends on the presence of a lesion in the heart producing emboli, and this is more common in young persons. Hemorrhage due to atheroma is an affection of advanced life. Apoplectic symptoms, such as coma, are more common in hemorrhage; but the distinction between these two states is often problematical. Sinus thrombosis or syphilitic endarteritis may cause a hemiplegia. Syphilitic hemiplegia cannot always be recognized unless from the history or the Wassermann test. If there are headache, involvement of cranial nerves, especially the third, and an irregular mode of onset, the diagnosis is much more probable. In a young adult the absence of a cardiac lesion, such as could cause embolism, is further suggestive of syphilis as the cause of the hemiplegia.

VII. THE CEREBRAL PALSIES OF CHILDREN.

Children are sometimes the victims of hemiplegia, diplegia, paraplegia, and speech defects, due to affections of the brain. As these conditions present some special features they demand special notice apart from similar affections in adults.

Pathology.—Cerebral hemorrhage is rare in children, unless in the case of accident or trauma, especially at birth. These birth palsies are usually due to meningeal hemorrhage, caused by a general asphyxia, which in its turn is caused by prolonged pressure on the placenta. This is indicated by the fact that hemorrhage in the new-born is sometimes observed beneath other serous membranes, as, for instance, the capsule of the liver. Hence intracranial hemorrhage is not necessarily caused by the forceps, although this may be a factor in some cases. Hemorrhage may also be caused by the

paroxysms of whooping-cough. A cerebral sclerosis occurs in young children; and Strümpell suggested that there may be also a polioencephalitis. Vascular lesions, as periarteritis and embolism, are doubtless the causes of extensive destructive changes in the brains of children, following upon the infectious diseases. It is not to be forgotten that hereditary, or even acquired, syphilis may cause cerebral palsies in children. Thrombosis of the cerebral veins may cause juvenile hemiplegia and diplegia. It occurs occasionally in typhoid fever, measles, etc. Thrombosis of the cranial sinuses is also occasionally observed. Hemorrhage is sometimes present beneath the membranes of the spinal cord.

A destructive lesion may result in the formation of a cavity in the cerebrum—the so-called porencephalus.

Symptoms.—The commonest forms of these palsies in children are hemiplegia, diplegia, and paraplegia.

The onset of the affection may be insidious, or at least not promptly recognized, as in very young children, and especially in the birth cases. When the onset is acute the affection may be ushered in with convulsions and coma, but this is by no means a universal rule. The "stroke" caused by embolus is sudden, just as in adults. Among other and minor symptoms are slight fever, vertigo, and vomiting. Confusion and delirium are sometimes seen.

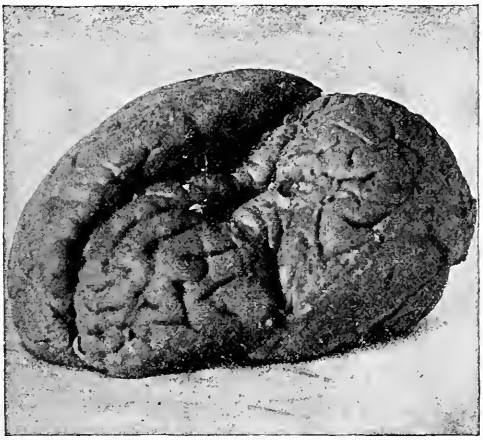


Fig. 369.—Porencephalus.—Lloyd.

Hemiplegia is the most common form. At first the paralysis is flaccid, but in a later stage contractures set in and the paralyzed limbs are much hampered and even deformed. They do not grow quite normally, but true muscular atrophy, as seen in spinal cases, is not present. The deep reflexes are exaggerated. In some cases athetosis is present. If the lesion is in the left cerebral hemisphere grave speech defects are present, but these differ somewhat from genuine aphasia, because if the lesion comes on in very early life before the child has learned to talk, the speech is undeveloped rather than impaired. The face in old standing cases is usually not paralyzed, and the arm is more paralyzed and contractured than the leg. As a rule there is no hemianæsthesia. The limbs may be cold and blue, but the reactions of degeneration are not present, and fibrillation is not seen. The gait is typically hemiplegic, and the arm is usually carried flexed and contractured at both the elbow and wrist. The bladder and bowel are not paralyzed.

Diplegia is simply a double hemiplegia, and is sometimes called bilateral spastic hemiplegia. The spasticity of the limbs is especially noticeable, not because the contractures are worse than in hemiplegia, but because, being on both sides, they give the patient a characteristic aspect. In one respect, however, the contractures in diplegia appear to differ from those of hemi-

plegia—they are somewhat more marked in the lower limbs. This gives the child a characteristic gait, if he is still able to walk. The limbs are usually adducted and extended, and the feet may be crossed and held in the position of equinovarus. The deep reflexes are exaggerated; sensation is unimpaired; and the upper limbs share in the rigidity. In these cases of double hemiplegia there may be imbecility or idiocy. Epilepsy and athetosis may complicate the case; speech defects are common; and strabismus and nystagmus are sometimes seen. The condition depends on a lesion which involves both hemispheres, and the destruction of brain tissue is sometimes great.

Fig. 370.—Old infantile hemiplegia.

Paraplegia of cerebral origin has been described only in recent years, and its pathology is still a matter of some obscurity. Some authors claim that it depends upon a limited brain lesion, in which the leg areas alone are involved, while others attribute it to a primary lateral sclerosis. It is practically identical with the spastic paralysis of the legs as seen in diplegia, but the arms are not involved. There may be epilepsy, athetosis, and idiocy, just as in the other forms of cerebral palsy in children. Paralysis of the bladder and bowel is not a necessary part of the symptom-complex, as in spinal paraplegia, but incontinence may result from the mental defects.

Monoplegia, either brachial or crural, is a rare form of cerebral palsy in children. In this form one arm or one leg alone is involved.

In this connection brief mention may be made of *Little's disease*. The affection is in fact a form of cerebral palsy, in which the motor-conducting

Fig. 371.—Congenital diplegia; early stage.—Young.

paths from the brain are injured, diseased, or undeveloped. Hence its clinical form is that of a diplegia or a paraplegia, according to the extent

of the injury. It occurs especially in children who are prematurely born. Some writers seem inclined to limit the term to mild cases in which the cerebral faculties are not much involved, convulsions are absent, and the tendency to improvement is marked, but there seems to be no good reason for retaining it as a designation for a distinct disease.[1]

FIG. 372.—Spastic diplegia; athetosis.—Lloyd.

The curious phenomenon known as "mirror-writing" is sometimes seen in these cases, especially in right-sided hemiplegia, when the child is taught to write with the *left*, or unparalyzed, hand. The writing is reversed, from right to left, so that it can only be read properly when held up to a mirror. Ireland and others have observed this thing in feeble-minded children who are left-handed.[2]

Hemianopsia and other changes in the visual fields have also been seen.

Sachs has described a condition which he calls *amaurotic family idiocy*, in which the child, soon after birth, becomes weak and lethargic; blindness, due to degenerative changes in the optic nerves, ensues; and spastic paralysis with increased tendon reflexes may be added. In some cases nystagmus, strabismus, and deafness are noted. There are no convulsions, but there is well-marked idiocy; and death occurs in early

FIG. 373.—Spastic diplegia; epilepsy; idiocy.—Lloyd.

childhood. Several cases have been observed in one family. The condition

[1] For an excellent account of Little's disease see Brissaud's Leçons sur les Maladies Nerveuses, Paris, 1895, p. 108.

[2] The Blot upon the Brain, Paper xii, p. 309.

is one of failure of development (agenesis) of the nerve-centres; its causation is obscure.

Diagnosis.—The diagnosis is, as a rule, not difficult. The spastic paralysis, usually hemiplegic or diplegic in type, with exaggerated reflexes, absence of muscular atrophy and electrical changes, and the associated mental defects, often with athetosis and epilepsy, distinguish these cases clearly from diseases of the spinal cord, especially anterior poliomyelitis. Gross lesion of the brain, such as tumor, might stimulate these cases, but the history and course are different, and optic neuritis is often present in the case of tumor.

Paraplegia of cerebral origin may simulate a spinal paraplegia, but in the latter there are no true cerebral symptoms, such as idiocy and epilepsy, and the bladder and bowel are almost always paralyzed. In case the lumbar cord is involved the paralysis is flaccid, with atrophy, lost knee-jerks, and even the reactions of degeneration. The history and course are also different. Infantile paralysis, due to acute anterior poliomyelitis, is usually confined to one limb; muscular atrophy occurs with lost knee-jerk and reactions of degeneration, and there are no cerebral symptoms. The onset is acute, with constitutional symptoms.

Obstetrical paralysis, especially of the brachial plexus, could hardly be confounded with a cerebral palsy. The paralysis is flaccid, the muscles waste, the deep reflexes are lost, electrical changes are present, and cerebral symptoms are wanting.

Fig. 374.—Attitude in cerebral palsy; paraplegic type.—Young.

The spastic rigidity of rickets and of tetany is distinguished by the associated symptoms of those diseases, the history, and the etiology.

VIII. HYDROCEPHALUS.

Pathology.—The lateral ventricles are enormously distended; the ependyma thickened; and the foramen of Monro or the aqueduct of Sylvius, or both, are possibly occluded. The brain may be so stretched as to be little more than a shell, the convolutions thin and flattened, and the sulci almost obliterated. The basal ganglia, the mid-brain, pons, medulla oblongata, and cerebellum are sometimes compressed and only partly developed. The choroid plexus may be thickened and congested. The bones of the skull are thin and translucent, and usually the sutures and fontanelles are widely distended, the former as much even as an inch. The essential elements,

or neurons, of the cortex suffer greatly, and the optic tracts and cranial nerves may be degenerated.

Etiology.—The causation is obscure. Some observers attribute the disease to occlusion of one or other of the natural foramina, such as the foramen of Monro, the aqueduct of Sylvius, or the foramen of Magendie. Not enough attention has been paid to the state of the choroid plexus and veins of Galen. Recently much has been written by Lees, Barlow, and others about a posterior basic meningitis, which causes occlusion of the foramen of Magendie, with consequent distention of the ventricles.

Symptoms.—Besides the distention of the skull there are seen various defects of development of the brain and nervous system. There may be mental impairment, ranging from slight imbecility to complete idiocy. In rare cases, however, there is preserved quite a remarkable mental integrity.

Fig. 375.—Hydrocephalus.—Lloyd.

The motor symptoms are often prominent; there is hemiplegia, diplegia, or monoplegia; the muscles are usually spastic, even contractured, and the deep reflexes are exaggerated. The eyes may be deflected downward, and there may be various forms of oculomotor palsy, with nystagmus. Other symptoms more or less common are convulsions, pain, as shown by the "hydrocephalic cry" (but this is more common in acute tuberculous meningitis), blindness, and incontinence. In extreme cases the child is bed-ridden from inability to hold up the enormously distended head. Occasionally hydrocephalus is associated with spina bifida.

Diagnosis.—This presents no difficulty in the advanced cases; the child's appearance is enough. In early stages, however, the diagnosis must rest on the child's evident failure properly to develop, and on the gradual enlargement of the head. Hydrocephalus sometimes begins before birth, and the skull may be greatly distended, causing grave dystocia.

IX. INTRACRANIAL ANEURISMS.

The larger aneurisms which develop on the main intracranial arteries, particularly at the base, will be discussed. The arteries usually involved are those that form the circle of Willis, and their branches, especially the middle cerebral. The internal carotid, cerebellar, and basilar arteries also are sometimes affected.

Pathology.—Diseases affecting the coats of the arteries, especially atheroma and syphilis, are the determining causes. Trauma also acts in this way. Emboli from vegetating endocarditis may be a cause. The aneurism is

either fusiform or sacculated: it is likely to increase rapidly, and it eventually bursts with fatal effect.

Symptoms.—The aneurism acts by compression like a brain tumor. Some authors describe a thrill or murmur audible on the skull or over the great vessels. The patient sometimes has a subjective sense of pulsation. Headache, vertigo, vomiting, and affections of consciousness occur. In some cases the aneurism is latent until rupture occurs, while in other cases the symptoms are paroxysmal, from successive small bleedings. Aneurism of the internal carotid may compress the optic nerve or optic tract, the nerves of the eyeball, and the first division of the fifth nerve, and as it increases may even cause hemiplegia, and, if it is on the left side, aphasia. Starr observed a case of left third nerve paralysis with right hemiplegia. The optic chiasm may be compressed by aneurism of the carotid and especially of the anterior communicating artery. Various affections of the retinal and visual fields thus result, and bilateral temporal hemianopsia has been reported. Aneurism of the anterior cerebral causes symptoms similar to those of the preceding, except when it is far to the front, when it may involve no cranial nerves and cause only obscure compression symptoms in the frontal lobes. Aneurism of the middle cerebral, especially if well within the Sylvian fissure, causes hemiplegia: possibly hemianæsthesia and hemianopsia: and, on the left side, aphasia. The third nerve may be compressed. There may be loss of smell on the affected side in the case of aneurism of any one of these arteries near the olfactory nerve. Aneurism of the posterior communicating artery may involve the optic tract and the third and sixth nerves: and if it grow very large, it may compress the peduncle, causing hemiplegia. Aneurism of the posterior cerebral may compress the peduncle and the third and sixth nerves, causing hemiplegia alternans: and if it should involve the occipital lobes it might cause various affections of the visual fields. Aneurisms of the basilar and vertebral arteries cause pontile and bulbar symptoms. The most striking is the compression bulbar palsy sometimes seen. If the pons alone is involved there may or may not be unilateral symptoms, such as hemiplegia alternans—paralysis of the sixth and seventh nerves on one side with opposite hemiplegia. If the medulla oblongata is involved the bulbar symptoms are dysarthria, dysphagia, paralysis of the tongue and lips, and sometimes respiratory symptoms, especially when the head is thrown forward.

Diagnosis.—Intracranial aneurism simulates a brain tumor, and the distinction between it and a neoplasm cannot always be made. A murmur, having the cardiac rhythm, controlled by pressure on the carotid, is suggestive of aneurism, but even this sign is not reliable, for such a murmur has been heard in other lesions. Starr claims to have heard a loud double murmur over the Sylvian region in a case of extensive softening. Murmurs in the head have also been heard in cases of tumor (when the growth is near a large artery), in anæmia, in hydrocephalus, in exophthalmic goitre, and in several cases of loud endocardial murmurs, which I have observed. There are therefore no positive rules for diagnosis. The history and course may be the same in aneurism as in tumor of the brain. Mills calls attention to pulsating exophthalmus as a sign of aneurism of the internal carotid.

X. TUMORS OF THE BRAIN.

Under this term are included all new growths within the cranium, whether within the brain, in the membranes, or springing from the bones of the skull. These tumors are comparatively rare, but in any large neurological clinic several of them are likely to be seen in the course of a year.

Pathology.—Intracranial tumors are of various kinds. In a series of 100 cases, analyzed by Mills and Lloyd, 16 were gliomata, 15 sarcomata, 13 gummata, 13 tuberculous, 7 carcinomata, 16 unclassified, and the remainder of various forms. An area of congestion, inflammation, softening, or hemorrhage is sometimes seen about the tumor; more rarely suppuration. The cerebrospinal fluid may be increased, and in some cases the ventricles are distended, this depending on the seat of the neoplasm. The tumor may be encapsulated, especially if it be a meningeal growth, in which case it is sometimes easily shelled out. If the tumor is within the substance of the brain, as, for instance, in one of the cerebral hemispheres, this may appear swollen, and even slightly flattened and discolored from pressure. Occasionally the cranial nerves are pressed upon or stretched over the surface of tumors at the base. In some cases the new growth closely resembles brain tissue. Tumors of the brain are sometimes multiple, and in the case of carcinomata they may be metastatic.

Symptoms.—The onset of a tumor is usually insidious, and the course gradual and even slow. The symptoms are general and local.

The *general* symptoms are such as are common to all kinds of brain tumors, and indicate in a more or less distinctive way the presence of an intracranial lesion. These symptoms are headache, vertigo, vomiting, convulsions, paralysis, ataxia, sensory changes, optic neuritis; and affections of consciousness. Headache is a very common symptom of brain tumor. In the early stages it may be slight, increasing later to great intensity. Sometimes it is paroxysmal; sometimes, but rarely, it is absent (in 5 out of the 100 tabulated cases); occasionally it is localized. It is not easily controlled by drugs. Vertigo, with which we may include affections of equilibration, forced movements, and ataxia, is seen in many cases. Some of these symptoms, such as affections of equilibration and forced movements, are highly characteristic of cerebellar tumor; but vertigo is not confined to subtentorial growths. Vomiting is usually of the propulsive kind, irrespective of food in the stomach; in other words, of the type known as cerebral. It is by no means constant, but when associated with other general symptoms it is highly suggestive. It may be unaccompanied with nausea. Convulsions occur in many cases; they are either general or focal. The former are seen in practically all kinds of cases; the latter are indicative, as a rule, of irritation of the motor centres in the cortex. By focal convulsion we mean one commencing in or confined to one or a few groups of muscles. Paralysis in some form is usually present, and it may or may not be an early symptom. It is more appropriately discussed among the local symptoms, as may also be said of the sensory changes. Optic neuritis is a frequent symptom, but it occurs also in other conditions, especially in brain abscess, purulent encephalitis, and brain syphilis. In brain tumor it occurs in at least 80 per cent. according to Gowers, Bramwell, and others. From another view-point

Oppenheim claims that of all cases of choked disk 90 per cent. are due to tumor of the brain. Affections of consciousness range all the way from slight stupor to profound coma. The emotions may be affected, and hysterical symptoms are sometimes seen.

The *local* symptoms are such as indicate the site of the tumor. The most important of these symptoms are the various forms of paralysis. Thus hemiplegia indicates that either the motor cortex or the descending motor tracts are involved. A monoplegia, as of the leg, arm, or face, is especially indicative of a lesion of the respective centre in the pre-Rolandic area. Aphasia points to involvement of the left cerebrum, in one or other speech centre, according to the type of the aphasia. Paralysis of the various cranial nerves, especially the third, fourth, fifth, sixth, seventh, eighth, and twelfth, is often of definite localizing value, as will be shown presently. An ataxic form of paralysis is sometimes seen in lesions of the superior parietal lobule. Affections of sensation may take the form of hemianæsthesia, hemianopsia, astereognosis, or localized anæsthesia, and will be considered with the focal diagnosis. Focal convulsions, of the type known as Jacksonian epilepsy, indicate usually a lesion in the respective motor centres. When associated with focal paralysis they are especially typical of a focal lesion in the motor area. It must be borne in mind that a convulsion may have a focal type at the beginning, passing later into a general convulsion; in such cases a focal origin of the convulsion is usually indicated, hence such fits have diagnostic value.

The *focal diagnosis* of tumors at various sites in the brain is briefly indicated as follows:

Tumors of the frontal lobe may give few if any localizing symptoms unless they involve the motor area. The mental changes are sometimes characteristic, and may consist of alterations of character, lack of power of attention, and especially retarded cerebration. If the motor area is involved, a tumor in the upper part of the pre-central gyrus, or on the mesial aspect of that region, causes paralysis of the opposite leg; in the middle part of the gyrus, paralysis of the arm; and in the lower part, paralysis of the face. On the left side a tumor involving the posterior part of the third frontal convolution causes motor aphasia.

The condition called perseveration, "lock-spasm," is sometimes seen in cases of tumor involving by pressure the motor area: thus the writer has seen a case of tumor on the mesial aspect of the brain, causing pressure on the arm centre, in which the patient's grip became so spasmodically fixed on his taking hold of an object, that he could not let go.

Tumors of the parietal lobe may also cause focal paralysis and convulsions by pressure on the pre-central gyrus; and if located on the left side they may cause motor aphasia for the same reason. Affections of the superior parietal lobule cause astereognosis and ataxic paralysis of the opposite leg or arm or both. If the posterior part of the internal capsule is involved there is hemianæsthesia, and possibly hemianopsia, on the opposite side. If the angular gyrus is involved there may be word-blindness. Tumors of the occipital lobe cause hemianopsia, possibly also hemianæsthesia; and also word-blindness, and object blindness. Tumors of the temporal lobe may cause hemiplegia and hemianæsthesia by pressure on the internal capsule,

if they are large enough, but the most typical symptom, if the tumor is on the left side and involves the first two temporal gyri, is word-deafness and verbal amnesia. Tumors of the mid-brain often cause a hemiplegia alternans, in which the hemiplegia is on the opposite side, while paralysis of the oculomotor nerve is on the side of the lesion—the so-called "syndrome of Weber." Tumors of the pons may also cause a hemiplegia alternans, in which with an opposite hemiplegia there is paralysis of the sixth and seventh nerves, possibly also of the fifth and eighth nerves, on the side of the tumor. Sometimes both sixth nerves are involved. These cranial nerve paralyses vary somewhat according to the site of the growth. Thus if the tumor is located high in the pons, above the decussation of the motor paths for these cranial nerves, the hemiplegia and the paralysis of the nerves are both on the opposite side. A favorite site for these tumors is in the cerebello-pontile angle. Cushing has described tumors of the acoustic nerve in this location, in which an early symptom is impairment of hearing, followed as the tumor grows with symptoms of pressure on other neighboring cranial nerves, as the sixth, seventh and fifth; also nystagmus and cerebellar symptoms.

A

FIG. 376A.—Paralysis of the sixth nerve of both sides and of the right seventh nerve, in a case of pontile tumor.—Lloyd.

Tumors of the cerebellum give a wide variety of symptoms, the most characteristic being loss of equilibration, cerebellar ataxia (in tumor of the vermis), and forced movements (in tumor of the peduncles, especially the middle peduncle). If the tumor presses upon the mid-brain, pons, or medulla oblongata, there may be characteristic paralysis of the several cranial nerves which have their nuclei in those structures, just as in tumors of those parts, and even hemiplegia. The knee-jerks may be lost, or may even go and come, but this symptom is not always seen. Tumors of the basal ganglia cause hemiplegia, hemianæsthesia, and hemianopsia, and on

B

FIG. 376B.—Paralysis of the seventh nerve, right side, and of both sixth nerves. Case of pontile tumor; patient attempting to close her eyes.—Lloyd.

the left side aphasia or paraphasia. Tumors of the membranes at the base of the brain, according to their location, cause paralysis of the various cranial nerves, especially the third, fourth, fifth, and sixth. Lloyd reported a case of total unilateral ophthalmoplegia, with anæsthesia of the first division of the fifth nerve, caused by a syphiloma just behind the orbit. Tumors of the medulla oblongata are rare; they cause paralysis of the twelfth nerve and of the motor tracts, and difficulty in swallowing and in respiration. In some cases brain tumor is latent, especially if it occupy a so-called *silent* region of the brain.

The Bárány tests of the labyrinth and vestibular nerve may give indications of the location of a subtentorial tumor. They require to be made by a competent otologist.

Tumors of the hypophysis, or pituitary body, give rise to two classes of symptoms; first, a dyscrasia, due to changes in the internal secretion of this body; second, to what are called "neighborhood," or pressure symptoms, due to the involvement of nearby cranial nerves. The dyscrasia may be the condition known as acromegaly (described elsewhere, p. 592) or it may be the *dystrophia adiposo genitalis*, in which the patient is undeveloped sexually, and is obese, with high tolerance for sugar, a juvenile appearance, and with possibly a polyuria. Gigantism is also seen. As the hypophysis is composed of several parts, with differences, as is believed, between their several secretions, these various dyscrasiæ are ascribed to alterations in these several secretions; but this subject is still in the hypothetical stage. The "neighborhood" symptoms are such as are caused by pressure on the optic chiasm (bitemporal hemianopsia, advancing to total blindness); oculomotor palsies, anosmia, etc. The X-ray may show changes in the sella turcica, especially the clinoid processes. There is a form of epilepsy sometimes seen in pituitary cases, the so-called uncinate type of fits, ushered in with a gustatory or olfactory aura, due to involvement of the neighboring uncinate gyrus.

FIG. 377.—Tumor of the cerebellum, showing forced movement to one side.—Lloyd.

The X-ray may also be useful in other brain tumors: as in a case of tumor of the pineal body, published by Lloyd. In that case there were calcareous deposits, giving a good shadow; but in most cases the X-ray plates require very cautious interpretation.

Diagnosis.—The above brief sketch sets forth the principles of local diagnosis; but brain tumors cause a wide variety of symptoms, both general and local, and these must be interpreted with care. A successful local diagnosis is frequently possible, and is often made.

Tumor may simulate abscess, but the history and course are usually

different; there is no history of a precedent focus of suppuration, there is no evidence of sepsis, the evolution is more gradual, and as a rule the duration is longer. Hemorrhage is not likely to be mistaken for tumor; the onset is entirely different. It is sudden, the symptoms are established quickly, and the case is not progressive. Yet a tumor in the motor region, causing hemiplegia, has been mistaken for a long-standing paralytic "stroke" due to hemorrhage. The history, the presence of optic neuritis and of headache in the case of tumor, should prevent error. Syphilis of the brain may closely simulate brain tumor, especially if the headache and optic neuritis are associated with focal symptoms, but the symptoms of syphilis often pursue an irregular course, quite unlike the steady progress of a tumor. Nevertheless, syphilis may cause a gummatous tumor. The laboratory tests for syphilis may help to clear up a doubtful diagnosis. In truth, tumor of the brain does not closely simulate any other lesion, unless it be brain abscess or syphilis; the onset, the course, the duration, and the grouping of symptoms are all sufficient as a rule to prevent error. It is well, however, in this connection not to overlook those curious cases in which hysterical symptoms have masked the symptoms of brain tumor and led to error.

XI. PARASITES IN THE BRAIN.

The commonest is the Cysticercus cellulosæ, the larval form of the Tænia solium, or pork tape-worm. Occasionally the echinococcus, or hydatid, is observed.

Pathology.—The parasites exist as cysts, of the size of a millet-seed to that of a grape or even a walnut. They may be found in the substance of the brain, or beneath the membranes, or floating free in the ventricles. The last is the most common. There is usually an ependymitis and great increase of the ventricular fluid. The parasites are found in both the lateral and the fourth ventricle.

Symptoms.—The nature of the disorder may be obscure. In a case observed by Lloyd[1] in the Philadelphia Hospital the earliest symptoms were apoplectiform attacks, followed by severe headache, hemiparesis, ataxia, disturbance of equilibration, exaggerated knee-jerks, incontinence of urine and fæces, drowsiness, loss of power of attention, speech defects, trismus, and failing vision. There were no convulsions nor optic neuritis. Death occurred in coma. At the autopsy eighteen cysts, some as large as a chestnut, clear and satin-like, were found floating in the right ventricle, which was enormously distended. One small cyst was found adherent in the fourth ventricle. There was ependymitis, and the aqueduct of Sylvius was occluded.

The patient in Lloyd's case insisted that when he moved his head he could feel something rolling within it.

The symptoms are not the same in all cases; much depends upon the number, size, and location of the cysts. Convulsions are not uncommon, and affections of consciousness are frequent. Headache is a constant symptom, and choked disk is sometimes observed. A cysticercus has been seen in the eye.[2] On the whole, the symptoms are irregular.

[1] Philadelphia Med. Journal, March 19, 1898.
[2] Oliver, Ophthalmoscopy, in Keating's Cyclopædia of the Diseases of Children, vol. iv, p. 238.

Diagnosis.—The diagnosis is most difficult, and the chances are that the true nature of the case will not be discovered until the autopsy. This is especially so in America, where the infection is rare.

Because of the irregularity of the symptoms, with headache, drowsiness, convulsions, and various forms of paralysis, the case may easily be mistaken for one of syphilis of the brain; also for brain tumor. There are no pathognomonic signs, and in any case the diagnosis must be problematical. The subjective sense of an object rolling in the head is the most distinctive sign that we have seen noted. It is quite impossible, as a rule, to trace the origin of the infection in the character of the patient's diet. There may be no tape-worm in the bowel, for autoinfection is not common, the eggs being usually introduced from without. If cysts are found under the retina or skin or in the muscles, the presence of similar growths in the brain is rendered highly probable.

XII. SYPHILIS OF THE BRAIN.

Syphilis is due to a living organism, the *Spirochæta pallida,* and some of its worst ravages occur in the nervous system. In the brain and spinal cord this organism begins its work, as a rule, in the perivascular lymph-spaces and upon the arteries. The inner and sometimes the outer coat of the vessel becomes infiltrated and thickened, as Heubner has pointed out. The inflammation then spreads to the meninges, and there results an exudative meningitis, which still further involves the blood-vessels and even invades the brain tissue and the cranial nerves.

Pathology.—There may be syphilis of the convexity, or of the base, or of both. At the base we often see the most typical picture of thickened membranes and gummatous infiltration. There is usually an associated cerebritis, especially near the surface, and deeper in there may be softening or hemorrhage from obstruction of the arteries or from rupture of their diseased walls. A common seat for this form of meningitis is between the cerebral peduncles and at the anterior perforated space, where the arteries run up to supply the interior of the brain, especially the lenticular nucleus and internal capsule. The basilar and vertebral arteries may also be involved. On the convexity there is seen sometimes a leptomeningitis or a pachymeningitis, or both. Occasionally the gummatous new growth forms a veritable tumor. It is thus easily understood how the syphilitic process causes damage to the brain by pressure, by inflammation, by sclerosis, by softening and hemorrhage, and by involving the cranial nerves. It also acts by elaborating secondary products—toxins.

Symptoms.—These may be exceedingly irregular, sometimes advancing quickly and then receding, or remaining for a long time stationary. From the character of the lesions it is evident that the symptoms may be multiform and show the invasion of many different parts of the brain.

Of general symptoms headache is common; it may be especially severe at night, causing insomnia. There may be vertigo and vomiting. Psychical changes are observed. There are drowsiness, stupor, confusion, dementia, even delirium and maniacal excitement. Convulsions, either general or focal, may occur in brain syphilis. The focal convulsions are usually

indicative of a lesion in some part of the motor area. When the motor area is invaded, as by a small patch of infiltration or sclerosis, there is focal paralysis as well as focal epilepsy. The cranial nerves at the base of the brain are often involved. Optic neuritis is seen, sometimes as an early symptom. Primary optic atrophy is not so common. The third nerve, one or all of its roots, is peculiarly exposed in the interpeduncular space to the action of the germ; next to it in frequency the sixth nerve, one or both. Ricord had a saying that syphilis puts its sign manual on the third nerve. Because of the involvement of the arteries, softening or hemorrhage results, especially in the lenticular nucleus and internal capsule, causing hemiplegia, hemianæsthesia, and aphasia.

The mid-brain may be involved, as shown by ophthalmoplegia and even by an alternate hemiplegia, in which the third nerve is involved on the side of the lesion, with hemiplegia of the opposite side. Pontile and bulbar symptoms result when the basilar artery is affected. There may be sixth or seventh nerve palsy on one side, with hemiplegia on the opposite side. Combined facial and trigeminal paralysis due to pontile syphilis has been noted. An apoplectic bulbar palsy, due to arterial disease, sometimes specific, has been described.

Pupillary changes are not uncommon in syphilis of the brain, especially loss of the reflex to light. Sometimes there is an internal ophthalmoplegia, *i.e.*, loss of the reflex to light and of the movement of the pupil on accommodation. Also irregular and unequal pupils.

Syphilitic tumors occur in various regions of the brain, and act like other tumors.

The symptoms of brain syphilis are often controlled by specific treatment, especially in the early stages, before destruction of tissue has occurred.

In some cases there are associated spinal symptoms—cerebrospinal syphilis; but the statement made by some writers, that it is an almost universal rule for both the brain and spinal cord to be involved in every given case, is too sweeping. Cases in which the disease is confined to the brain, or to the cord, as the case may be, are not so very uncommon. Nevertheless, careful search should be made for all symptoms.

Diagnosis.—The mode of onset and progress may suggest syphilis. The symptoms are often insidious, advancing irregularly, receding, then again advancing. This is seen in syphilitic hemiplegia. There may be slight apoplectiform attacks, then slight hemiparesis, then improvement, then a more grave attack, and so on. This can readily be understood from the nature of the syphilitic meningitis and endarteritis, interfering with circulation. The third nerve palsy is also sometimes irregular; only one or two branches may be involved at first, then later the whole nerve, due to the gradual involvement of the several roots of this nerve in the interpeduncular space. The history of the case is often clear, but sometimes it is not reliable. The multiformity of symptoms is often characteristic, and the presence of spinal lesions is generally conclusive. It may be difficult to distinguish syphilis in some cases from brain tumor, and often impossible to say whether a tumor is syphilitic or not. Brain tumors, as a rule, are more focal and constant in their symptomatology, and more regu-

larly progressive. The Wassermann test should always be made, as well as the laboratory tests for globulin, and a cell count for lymphocytosis, which is very common in nerve syphilis. This subject is discussed elsewhere in this work (p. 645, Vol. I). Tuberculous meningitis has a more rapid course than syphilitic meningitis; it is not amenable to drugs, and is uniformly fatal. There is also fever and slowing of the pulse, and the disease is commonly seen in children, whereas the syphilitic form is more common in adults. A febrile reaction is sometimes, though rarely, seen in brain syphilis. The hemiplegia of syphilis cannot always be easily distinguished from hemiplegia due to other causes. If there is a clear history of syphilis, no endocardial lesion to cause embolism, and the accident occurs in a young adult; and if, especially, there be headache, involvement of one or other cranial nerve, particularly the third, and the symptoms are of rather irregular onset and course, the evidence is in favor of syphilis. The laboratory tests of the blood and cerebrospinal fluid are important, but they should not be allowed to determine the diagnosis as against the clear indications of the clinic. Fournier claims that 90 per cent. of hemiplegias occurring in adults under 30 years of age are syphilitic.

XIII. GENERAL PARESIS.

General paresis is a syphilitic disease of the brain cortex, medullary tracts, and nerves. The disease occurs more frequently in men than in women; and in the white than in the colored races. It is said to be more common in civilized countries and among the better classes, and Krafft-Ebing's statement that it is always due to "civilization and syphilization" is probably based upon this opinion. Since Noguchi announced in 1913 that he had found the spirochæte in 48 brains among 200 cases of general paresis, its syphilitic origin has been accepted as demonstrated beyond doubt. Noguchi's results have since been confirmed by others, and the laboratory tests are still further confirmatory.

Pathology.—There is a destructive process in the tissues of the brain. This process shows itself in the small vessels of the membranes, leading to congestion, obstruction of the circulation and the lymph stream, morbid increase of the neuroglia, hardening and atrophy of the cortex, thickening of the membranes, minute hemorrhages, extravasation of the cerebrospinal fluid, distention of the ventricles, ependymitis, and impaired nutrition and destruction of the neurons in the brain cortex.[1]

According to Orton the vessels are the path by which the spirochætes reach the brain: others believe it is by the lymph-spaces. There is a widespread peri-arterial infiltration of lymphocytes and plasma cells—the characteristic of the syphilitic process.

Symptoms.—General paresis is usually divided into four stages.

In the first stage the prodromes appear, such as change of character, spells of irritability or even of depression, inattention to business, erratic conduct, and moral lapses. Insomnia, or its opposite, somnolence, may occur. Alcoholic and sexual excesses are common.

In the second stage the period of grandiose delusions sets in. The

[1] Bevan Lewis, Mental Diseases, 2d edit., p. 548; Berkley, Mental Diseases, p. 197. These two works give the best accounts of the morbid anatomy of general paresis.

patient has most extravagant ideas of his wealth, of his personal importance, even of his sexual prowess. He becomes talkative, boastful, and slightly demented. Along with these mental changes appear physical changes. There is tremor of the muscles of the face, tongue, hands, and limbs; speech becomes drawling, stammering, or staccato; the handwriting is unsteady, and words are dropped or run together just as in speech. The gait is weakened and incoördinate, with increased knee-jerks as a rule. There may be myosis, or irregularity and inequality of the pupils, and sometimes the Argyll-Robertson symptom, in which the light reflex is lost while the movement on accommodation remains. There may also be optic atrophy.

In the third stage the disease assumes a more chronic aspect, and dementia is more advanced. In this stage especially we see crises of epilepsy, apoplexy, hemiplegia, or maniacal excitement. The paralytic symptoms increase.

In the fourth stage the patient is in terminal dementia, paralyzed, bedridden, almost or quite speechless, with incontinence of urine and fæces—a total wreck, until death ends the scene in exhaustion or in an epileptic or apoplectic crisis.

In rare cases arthropathies form, especially when the disease is associated with posterior sclerosis of the spinal cord. This association with posterior sclerosis constitutes the disease known as taboparesis, in which many, or most, of the symptoms of locomotor ataxia are present with those of paresis.

In some cases remissions occur, of quite long duration, but the hopes raised by them are fallacious, for the disease is fatal.

Diagnosis.—This may present some difficulty in the early stages, as between this disease and disseminated syphilis of the brain. But in the latter there is not seen the characteristic psychosis with grandiose delusions, and the disease does not present the regular evolution that is seen in general paresis. Still, the diagnosis is not always easy. The therapeutic test may help to solve the problem, but it must be borne in mind that general paretics do not bear well the heroic antisyphilitic drugging. Striking examples of cerebral lues, resembling the early stages of general paresis, sometimes make good recoveries under a judicious treatment. These are probably the kind of cases that are reported as cures of general paresis. The Argyll-Robertson pupil would point to the latter disease.

There is a pseudoparesis induced by alcohol and promoted by morphia, cocaine, etc., which closely resembles the genuine disease, especially in the tremor and speech defects, but recovery occurs promptly on the withdrawal of the poison. The history in these cases is suggestive, and they do not present quite such a typical expansive psychosis as in general paresis; the mental condition is usually one of enfeeblement with hallucinatory delirium; which, however, may be rather grandiose.

The distinction between neurasthenia and the early stages of paresis is not difficult. The Wassermann and other laboratory tests should be made.

Multiple sclerosis differs from general paresis in the history of the case, in the nystagmus and intention tremor, and in the absence of the expansive psychosis.

XIV. SENILE DEGENERATION.

The fundamental change in the central nervous system in old age is probably atheroma of the blood-vessels. It is a trite saying that a man's life is only as long as the life of his arteries. Some of the effects of this arterial degeneration have already been discussed in the chapters on cerebral hemorrhage and softening. There are other conditions, however, so identified with senility that they merit especial, though brief, attention. Whether or not they all depend on changes in the blood-vessels may still be an open question.

FIG. 378.—Arthropathy in general paresis.—Lloyd, Philadelphia Hospital Reports, vol. ii.

CEREBRAL SYMPTOMS.—The most conspicuous of these are undoubtedly the mental changes which occur in old age. There is loss of memory and failure of the mental powers so familiar to all. The various psychoses of the aged, such as senile melancholia, delusional insanity, maniacal episodes, and dementia, belong rather to psychiatry than to clinical medicine.

The early symptoms of atheroma of the cerebral arteries are not easily recognized. Among them are tinnitus, vertigo, throbbing or fulness in the head, transient attacks of mental confusion, slight paresis, aphasia, and headache. The throbbing or beating in the head is sometimes almost enough to suggest aneurism, especially when accompanied by headache. It may even depend in some cases on slight aneurismal dilatations. We must not overlook in these cases associated disorders in the heart and kidneys.

SENILE EPILEPSY.—The onset of epilepsy in old age is occasionally observed. It is doubtless helped on in some cases by alcoholism. The prime cause is probably degenerative change in the brain cortex. The epileptic seizure in senile patients is practically like that seen in ordinary epilepsy. It is necessary to exclude uræmia.

SENILE TREMOR.—In some old persons a very pronounced tremor is seen. It usually begins as a fine movement in the hands, most marked on exertion. Later it may spread, especially to the neck, causing a shaking of the head. In advanced stages it is even present during rest, but it disappears during sleep. It is not accompanied with the muscular rigidity and the characteristic attitude, expression, and gait of paralysis agitans, although intermediate cases are seen, and the two conditions have some points of contact.

Senile Paraplegia.—A spastic paresis of the legs, more rarely of the arms also, occurs in the aged. There may, or may not, be anæsthesia and weakness of the sphincters. The cases usually resemble primary lateral sclerosis, without loss of sensation or involvement of the bladder and bowel. This condition may depend on primary changes in the cord, especially in the lateral tracts, or it is possibly due to small foci of softening in the motor regions of the brain. These patients often have well-marked mental deterioration.

Alcoholism and Drug Habits in the Aged.—Evil habits creep on insidiously in some old people who may always have led strictly temperate lives. Bevan Lewis calls attention to the frightful impetus which the excessive use of alcohol lends to the retrograde changes which naturally occur in the brain in old age. The opium habit may be formed by the aged.

XV. ACUTE DELIRIUM.

This disease was first described by Luther Bell, an American alienist, and is sometimes called from him Bell's mania.[1] It is also called typhomania and *delirium grave*.

Pathology.—The disease is probably an acute infection, and may be caused by a variety of germs. Bacteriological studies have not led to uniform results. Berkley says that in a malady which may be caused by so many agents, a sole cause is not to be expected.

Symptoms.—The onset is usually abrupt and the course very rapid. Delirium sets in acutely and advances quickly to stupor and coma. The tongue becomes dry and brown, and sordes form. The pulse is rapid and compressible. There is muscular unsteadiness, tremor, and incoördination. The temperature rises but pursues no regular course. There is aversion to food, and the vital powers soon fail. Death may occur in a few days. Cases of longer duration are seen to follow the puerperium, and are probably due to sepsis; but in the typical cases no cause can be made out.

Diagnosis.—The diagnosis rests upon the abrupt onset, the rapid course, and the tendency to speedy death without obvious cause. Bacteriological studies should be made. Perhaps with our increasing knowledge of microbian pathology the nature of these cases will be made clear. The possibility of poisoning by alcohol, syphilis, lead, or malaria must not be ignored. Fulminating attacks of typhoid fever, measles, and scarlatina may simulate *delirium grave*, but can usually be recognized by the associated symptoms. Uræmia must also be excluded.

XVI. MULTIPLE SCLEROSIS.

This disease, also called insular, or disseminated, sclerosis, is marked by foci of degeneration scattered through the brain and spinal cord.

Pathology.—The foci vary in size from that of a small bird-shot to that of a pea or a chestnut, and there may be even larger areas involved. They are different in color from, and harder in consistence than, the brain

[1] Bell described the disease at a meeting of the Association of Superintendents of American Asylums, in 1849.

tissue. Histologically they consist of hardened connective tissue and infiltrated blood-vessels, with degenerated nerve-fibres, although many fibres are seen intact, penetrating the diseased tissue—a fact which is supposed to explain one of the chief symptoms, the intention tremor. The cause of multiple sclerosis is not known; it is not believed to be syphilis, although diffuse syphilitic lesions sometimes cause a state which clinically is not very unlike multiple sclerosis. The fact that the disease sometimes follows the infectious diseases, such as smallpox, typhoid fever, etc., does not explain its causation. The same may be said of its appearance in metal workers.

Symptoms.—The affection is one of early adult life; it rarely appears after the thirtieth year, and it is not uncommon in young women.

There are three symptoms which especially distinguish insular sclerosis —intention tremor, scanning speech, and nystagmus.

The intention tremor is an early symptom. As its name implies, it appears on voluntary motion; the arm, for instance, showing wide jerky tremors when the patient attempts to use it, as for carrying a glass of water to the lips. The motion is then so violent that often a large part of the water is spilled. The tremor is coarse, with wide amplitude and few vibrations to the second. While the patient is at rest, it is absent. The tremor extends to the face, tongue, and limbs, causing other symptoms, especially scanning speech and an unsteady gait. Sometimes the head shakes.

The speech is usually scanning rather than staccato, although in all cases the words are uttered slowly, and sometimes with pauses between them. In a few rare cases bulbar symptoms have been seen, such as paralysis and wasting of the tongue. Pseudobulbar palsy has also been noted.

The nystagmus is usually a prominent symptom, and is most marked when the patient turns the eyeballs to one side—lateral nystagmus. Sometimes a rotary nystagmus is seen, in which the eyeballs are rolled on their axes. Even when the eyes are fixed straight ahead slight oscillations are sometimes seen.

The gait is usually spastic, and the deep reflexes are exaggerated in consequence of involvement of the lateral tracts in the insular foci at various levels. Abolition of the cremasteric reflex is claimed by Collins. The bladder and bowel are not paralyzed; if there are exceptions to this rule, they must be very rare.

In advanced stages the mental faculties may suffer, and crises of an apoplectiform kind may be seen. Psychoses, such as melancholia and dementia, may even occur; and a grandiose delusional state, not unlike that seen in paresis, is sometimes, though rarely, observed. Optic atrophy is present in some cases. Gowers claims that even when the optic nerve is involved in a patch of sclerosis many fibres pass through unharmed and a fair degree of vision is retained; but the visual fields are variously affected. One optic nerve may be more injured than the other. Uhthoff, who analyzed 100 cases of multiple sclerosis, found the optic nerves affected in 40. He also found paralysis in one or other of the ocular muscles in 17 of his cases. Involvement of the pupils was rare. Muscular atrophy sometimes occurs, and a slight ataxia.

Sensory symptoms are usually remarkable for their absence. Pain is sometimes felt, and various but slight modes of anæsthesia are sometimes present and are most likely to be found in the distal parts of the limbs.

The course of multiple sclerosis is chronic; remissions occur, and even slight improvement, but the disease is incurable.

Diagnosis.—The diagnosis is easily made from the association of the three cardinal symptoms. Nystagmus and scanning speech are seen in Friedreich's disease, but they are then associated with ataxia and lost knee-jerks, and the disease is usually a familial one. In disseminated syphilis the course is more rapid, the mind more involved, the evolution of symptoms is not characteristic, and scanning speech and nystagmus are not seen as a rule. General paresis shows scanning speech, tremor, and ocular changes, but the expansive psychosis, the history, the evolution, the more marked tremor of the facial muscles, all serve to distinguish it. The cremasteric reflex is likely to be preserved, but we do not insist here upon this sign as distinctive. The early diagnosis is uncertain.

Multiple sclerosis may be simulated by hysteria, in which, however, symptoms that can only be accounted for by organic lesions, such as nystagmus and optic atrophy, are never found. Cases of hysterical pseudo-sclerosis are usually of traumatic origin. The differential diagnosis is most important. Other hysterical stigmata are not always present. Somewhat similar cases follow exposure to mercury or lead. Chronic alcoholic tremor, especially in young adults, may present a rather confusing picture, but in these cases nystagmus and the characteristic speech are not present, nor is a spastic gait, with exaggerated deep reflexes, observed. The history, of course, is important.

XVII. DISEASES OF THE MID-BRAIN.

The mid-brain is composed in part of the cerebral peduncles, which contain the motor tracts from the cerebrum; its dorsal part consists of the corpora quadrigemina, and it is penetrated by the aqueduct of Sylvius, underneath which are located the nuclei of the third and fourth nerves. The sensory tract, or fillet, runs up just behind each peduncle. The red nucleus is located in the mid-brain.

Pathology.—Tumors are sometimes observed in this region, and more rarely hemorrhage and softening. Wernicke has described an acute destructive process located in the gray matter about the aqueduct, which he has named *superior polioencephalitis*. The meninges in the interpeduncular space are not infrequently the seat of syphilitic meningitis.

Symptoms.—Tumors of the mid-brain are usually unilateral, and cause hemiplegia alternans, in which there is an opposite hemiplegia, with or without hemianæsthesia, associated with paralysis of the third nerve on the side of the lesion. Other symptoms of tumor are usually present, such as optic neuritis, headache, vertigo, vomiting, changes in consciousness, and more rarely convulsions. A somewhat similar train of symptoms may be caused by cerebellar tumors, if these make pressure on the mid-brain; but in cerebellar tumors there is likely to be in addition some disorder of

equilibration. A meningeal tumor in the interpeduncular space may paralyze one or both third nerves.

In Wernicke's acute superior polioencephalitis there is a destructive process in the mid-brain, and sometimes in the gray matter of the third ventricle. The floor of the aqueduct of Sylvius, hence the oculomotor nuclei, and even the peduncles, are involved. The symptoms are paralysis of the third and fourth nerves, nystagmus, optic neuritis, and rapid prostration, sometimes with ataxia, dysarthria, and even paralysis of the face and extremities. Death is common in from eight to fourteen days.

Hemorrhage and softening in the mid-brain are rare. The symptoms are those of a focal lesion, such as ophthalmoplegia of various kinds, according to the nuclei involved, hemiplegia alternans, etc.

Diagnosis.—This rests upon the grouping of symptoms as described above. The most characteristic is the hemiplegia alternans, in which the third nerve is paralyzed on the side of the lesion and the hemiplegia is on the opposite side. In Wernicke's disease the association of symptoms and the rapidly acute course, often with fatal ending, are characteristic. The disease is to be distinguished from bulbar palsy by the history and course and especially by the different cranial nerves involved. In the mid-brain lesion the eyes are paralyzed; in the bulbar lesion, the tongue and lips.

Interpeduncular syphilitic meningitis is distinguished by the headache, the third nerve palsy, the irregular course, the absence usually of hemiplegia, and the history. There may, however, be hemiplegia if either peduncle is softened by syphilitic endarteritis. Complete paralysis of both third nerves is not common; in fact, the third nerve palsy may change from time to time, and it is usually unilateral.

XVIII. NUCLEAR OPHTHALMOPLEGIA.

By this term is meant an affection in which the muscles of the eyeballs and upper lids are paralyzed by reason of disease of the nuclei of their motor nerves. The disease is often chronic and selective, for it picks out gradually the nuclei of the third and fourth nerves, which are in the mid-brain, and those of the sixth nerves, which are in the pons, some distance away. Consequently it is neither a purely mid-brain nor a purely pontile disease.

Hutchinson was one of the first to describe a pure nuclear ophthalmoplegia. Later Wernicke described an acute destructive process invading the floor of the aqueduct of Sylvius and neighboring parts in the mid-brain, which he called *superior polioencephalitis* to distinguish it from bulbar disease, which he called *inferior polioencephalitis:* but cases of this affection often present other than purely nuclear ophthalmoplegic symptoms, as, for instance, optic neuritis, nystagmus, facial paresis, dysarthria, ataxia, and even hemiplegia; in other words, it is not a purely nuclear disease. It is best to limit the description, therefore, to the nuclear disease, a sufficient number of cases of which have now been reported to entitle it to distinction as a substantive affection.

Pathology.—The disease process has some resemblance to the chronic

or subacute forms of anterior poliomyelitis, inasmuch as the multipolar ganglion cells are gradually destroyed. From this form there are all grades, apparently, up to the highly acute types in which the process is more wide-spread and the case may end fatally in a few days.

Syphilis is doubtless the cause in most cases, as was pointed out by Hutchinson; and the mode of onset may be by invasion of the walls of the vessels supplying the nuclei of the nerves.

Symptoms.—Brissaud has proposed a useful classification of the ophthalmoplegias as follows: The affection is *total* if all the muscles of the eyes, both exterior and interior, are involved; *partial,* if only some muscles are paralyzed; *complete,* if the paralysis in the affected muscles is absolute; and *incomplete,* if the affected muscles are not absolutely paralyzed, but only paretic. Hutchinson describes an ophthalmoplegia *externa* in which the interior muscles, that is, the iris and ciliary muscle, escape; and the opposite form is the opthalmoplegia *interna* in which only the iris and ciliary muscle are paralyzed. This is possible, because the nuclei for the iris and ciliary body lie some distance anterior to the other nuclei. Finally, ophthalmoplegia may be either unilateral or bilateral, but the unilateral cases are never nuclear, as will be explained later. All these nuclei, except the sixth, lie underneath the aqueduct of Sylvius; those for the iris and ciliary body, however, lie somewhat farther forward, even in the walls of the third ventricle.

Fig. 379.—Nuclear ophthalmoplegia.—Philadelphia Hospital—Lloyd.

In a case, studied by Lloyd, the patient, a woman aged 35, noticed first external strabismus in the right eye, then ptosis, then external strabismus in the left eye, and finally, after some months, loss of all ocular movements except in the left external rectus. This patient had headache and abolished knee-jerks, but no fulgurant pains and no ataxia. There was possibly some beginning optic atrophy. No history of syphilis was obtainable.

In most of the described cases this gradual progress has been noted, significant of a slowly progressive nuclear disease. First one ocular muscle and then another, in one eye and then in the other, becomes paralyzed until all, or nearly all, are involved. Various forms of strabismus occur, until the eyeballs become motionless and ptosis is complete. It is occasionally seen in young persons, even in children, and Möbius called it then "infantile nuclear atrophy."

Diagnosis.—Syphilitic meningitis between the cerebral peduncles may involve the roots of the third nerves, and may possibly, but not probably, extend to the sixth. Headache is present, and possibly optic neuritis, but the resemblance to nuclear disease may be striking. Syphilis of the third nerve is often unilateral. Moreover, in interpeduncular syphilis the

paralysis is confined to the third nerve; the fourth and sixth escape. However, the question whether the syphilitic disease in these cases is purely and primarily nuclear, or whether it is primarily in the meninges, nerve-roots, or blood-vessels, is still an open one.

Tumor of the mid-brain does not invade the nuclei alone; it usually causes paralysis of the limbs, sometimes a hemiplegia alternans, that is, paralysis of the third nerve on the side of the lesion with opposite hemiplegia; also optic neuritis, headache, and pressure symptoms. This may be true, also, of extensive syphilitic disease.

Wernicke's acute superior polioencephalitis is known by its history, its rapid course, its invasion of other than nuclear territory, and its consequent wider range of symptoms.

Nuclear ophthalmoplegia, rarely or never total, occurs in locomotor ataxia, but it is not usually an early symptom, and it is associated with true tabetic symptoms, such as ataxia, fulgurant pains, optic atrophy, lost knee-jerks, etc. The commonest form is bilateral ptosis. Some observers claim, however, that a chronic progressive ophthalmoplegia is sometimes a precursor of tabes dorsalis, combined sclerosis, multiple sclerosis, or even progressive muscular atrophy. Hence a cautious diagnosis is called for, since it may take months or even years to determine the question. In late years it has been disputed whether these tabetic ophthalmoplegias are ever really nuclear in origin, and not rather meningeal or radicular.

FIG. 380.—Nuclear ophthalmoplegia. Upper lids supported in order to show the position of the eyeballs.—Philadelphia Hospital—Lloyd.

Unilateral ophthalmoplegia, total and complete, is never nuclear. The one-sided cases are usually caused by some local lesion at the base of the brain. The first division of the fifth nerve is likely to be involved in these basilar cases; hence there is anæsthesia of the conjunctiva and brow on one side; and if the second and third divisions of the fifth nerve are involved, the anæsthesia extends over one-half of the face and tongue. These cases should not be mistaken for isolated paralysis of individual nerves, in which the loss of power is confined to the nerve affected. The third or the sixth nerve on one side is occasionally paralyzed by trauma.

PARALYSIS OF ASSOCIATED MOVEMENTS OF THE EYES.—These are the lateral, the upward, and the downward movements, and the movement of convergence. The evidence is in favor of a centre in the brain cortex for associated ocular movements. Grünbaum and Sherrington found a centre for lateral movements of the head and eyes in the frontal lobe somewhat apart from the rest of the motor area, and Ferrier has shown from many recorded instances that conjugate deviation of the head and eyes is caused by a lesion in the posterior end of the middle frontal gyrus. Perinaud,

Mott, and others hold that this ocular centre is subdivided for the various associated movements; thus, one part is for the lateral, another for the upward, and still another for the downward movements. As a fact, however, it is the conjugate lateral movement which is usually affected, and this is seen in a variety of lesions, as, for instance, in large cerebral hemorrhage, and in thrombic and embolic softening. Any lesion which affects this centre directly or cuts off its underlying connections may cause conjugate lateral deviation of the head and eyes. The patient looks *toward* the side of the lesion when the paralysis is complete; but if the lesion is an irritative one, as in focal or one-sided epilepsy, he looks *away* from the lesion. The lateral deviation of the eyes that is caused by a cerebral lesion is usually temporary, and it is associated with lateral deviation of the head. It is thus distinguished from the lateral deviation of the eyes which is sometimes seen in lesions in the pons, located in the posterior longitudinal bundle, by which the nuclei of the third, fourth and sixth nerves are joined. In these pontile cases the paralysis is not transient, it is not likely to be associated with deviation of the head, it may be accompanied with other pontile symptoms, and the deviation is sometimes, but not always, *away* from the side of the lesion.

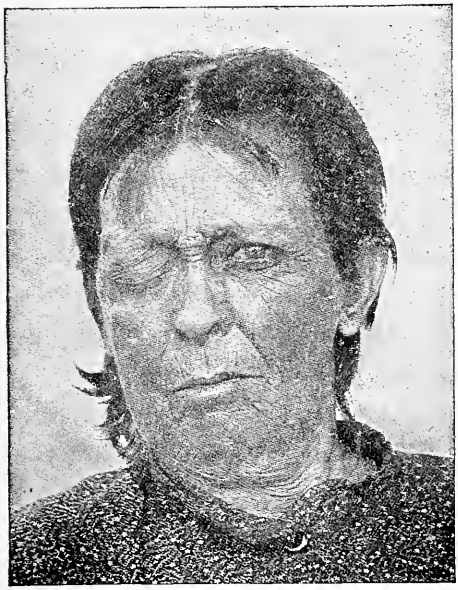


Fig. 381.—A case of unilateral ophthalmoplegia.—Lloyd.

Paralysis of the associated upward movement of the eyes is sometimes seen, and is sometimes accompanied with paralysis of convergence. The associated downward movements may be paralyzed, but this isolated paralysis is rare; it is usually associated with paralysis of the upward movements. The lesion in these cases is located on or near the floor of the aqueduct of Sylvius, and it may be a tumor, a syphilitic inflammation, or a spot of softening. It is possible, however, that paralysis of associated upward or downward movements may be caused by a lesion cutting off the cortical centres from the nuclei in the mid-brain. Wernicke has called this condition pseudo-ophthalmoplegia, and Lloyd has recently reported a case of pseudobulbar palsy, due to bilateral lesions in the lenticular nuclei, in which there was loss of power in the associated upward movements of the eyes.

Marie claims that paralysis of associated ocular movements may be caused by hysteria; but in such a case the affection would more likely be a spasm of the opposing muscles than a true paralysis. Thus a spasm of both superior recti muscles, pulling both eyes upward, would present the appearance of a paralysis of the inferior recti and the superior oblique muscles, whose function it is to pull the eyes downward. The patient would

probably present other hysterical symptoms. Gilles de la Tourette thinks that hysterical paralysis of any of the eye muscles is rare, and that the real affection is a spasm or contraction of the opposing muscles. There is, for instance, an hysterical blepharospasm which may simulate a bilateral ptosis.

The muscles of accommodation (ciliary) and of convergence (internal rectus) usually act together, and in both eyes at the same time. Thus vision is accommodated for near objects. But the conjoint action of the two internal recti for convergence may be interfered with, and convergence may be paralyzed while these two muscles continue to act in other associated movements. Perlia has described a special nucleus for convergence beneath the middle line of the aqueduct of Sylvius—called Perlia's *central nucleus*. A destructive lesion at this point presumably causes isolated paralysis of convergence; that is, the internal recti muscles fail to turn the eyeballs inward in attempts at convergence, but they may act properly in associated lateral movements to the right or left.

In determining these various paralyses of associated movements it must be borne in mind that the eyeballs roll *away* from the paralyzed muscles: thus, if the upward movement is paralyzed the eyeballs roll downward, and *vice versa*. If there is right conjugate lateral deviation, the muscles paralyzed are the left external rectus and the right internal rectus.

XIX. DISEASES OF THE CEREBELLUM.

The superior cerebellar peduncles after decussating connect the cerebellum with the red nuclei in the mid-brain and hence probably with the cerebral cortex: the middle peduncles connect apparently with nuclei in the pons; and the inferior peduncles carry sensory neurons from the direct cerebellar tract of the spinal cord and also vestibulocerebellar fibres.

Tumor, abscess, and other focal lesions of the cerebellum have been considered under a previous heading. Besides tumor and abscess there are occasionally seen softening and terminal cysts in the cerebellum, due to occlusion of the blood-vessels, as in syphilis and atheroma. Extensive softening of one cerebellar hemisphere may occur with little or no evidence of cerebellar disease. Atrophy and sclerosis of the cerebellum are also seen in cases of arrest of development, and in the cerebellar form of hereditary ataxia.

Symptoms.—In gross lesions of the cerebellum incoördination and forced movements are often seen. There may be wide staggering and swaying, the patient standing with the legs far apart, or there may be reeling and even rotary movements. These symptoms are attributed to lesions of the middle lobe. Forced movements are supposed to depend rather on lesions of the middle cerebellar peduncles. They may be so aggravated that the patient cannot even sit upright in bed, but is forced to one side. Paralysis of the oculomotor nuclei is sometimes caused by pressure on the mid-brain; and pontile symptoms, such as paralysis of the fifth, sixth, seventh, and eighth nerves, and even hemiplegia, are also caused by gross lesions of the cerebellum pressing on the pons. Optic neuritis or atrophy is common, and headache, vomiting, and vertigo are seen just as in other intracranial affections. When one cerebral hemi-

sphere is alone affected there may be few if any symptoms, but this is not a universal rule. The knee-jerks are variously affected. In some cases of tumor or abscess they disappear, and even return again, or they are merely exaggerated or diminished, as the case may be, or they are not the same on both sides. The functions of the cerebellum are still obscure, but they evidently are largely concerned with equilibration, as is proved by the diseases of the organ.

In case of a suspected tumor or other gross lesion of the cerebellum, pons, or cerebellopontile angle, the Bárány tests of the labyrinth and vestibular nerves should be made, preferably by an otologist specially skilled in the work. These tests give data for localization, but their entire reliability may still be open to some question. At any rate, they require skill in their performance and great caution in their interpretation.[1] G. W. Mackenzie, of Philadelphia, recommends a galvanic test.[2]

A great elaboration of cerebellar symptoms has recently been undertaken, based upon the phenomena of asynergy, hypermetry, adiodokocinesis and tremor. Asynergy is shown by a mismeasurement of movements, giving dysmetry or hypermetry. Adiodokocinesis is shown by the impossibility of correctly and rapidly pronating and supinating the hand. Tremor is shown by the finger-to-nose test. Mills and Weisenberg[3] have made a kinematographic study of these symptoms and have endeavored to show their localizing value in cerebellar cases. The subject is rather too complex and involved to admit of full discussion in these pages.

Cerebellar hereditary ataxia is an affection nearly allied to the spinal hereditary ataxia of Friedreich, but there are well-marked differences between the two. It develops according to most authors rather later in life, but has in some cases dated from a very early period, if indeed it were not congenital. In addition to ataxia there are speech defects, not mere scanning but rather an incoördinate and explosive type of speech; exaggerated knee-jerks; and optic atrophy. Nystagmus is occasionally seen. The disease may be familial, as in a remarkable series of cases reported by Sanger Brown. Lloyd is convinced that there are different types of cerebellar ataxia; that not all are necessarily familial or hereditary; and that optic atrophy is not always present. The most typical symptoms seem to be the ataxia, which is not always so extreme as in Friedreich's disease, and the incoördinate explosive speech. The knee-jerks are probably preserved or even increased in most cases. Some of these cases may be due to injury at birth. Some form of atrophy of the cerebellum has usually been found after death, but again the findings have been practically negative. The cerebellar tracts in the cord were involved in one of Brown's cases.

Diagnosis.—The diagnosis between Friedreich's ataxia and the cerebellar hereditary ataxia rests upon the difference in the speech defects, and upon the preservation or even increase of the knee-jerks, and possibly the presence of optic atrophy in the latter form. In chorea there is not a

[1] See paper on Tests by Bárány Methods, by Mills and Jones. Jour. Am. Med. Ass'n, vol. lxvii, pp. 1298-1300. Also a paper by Randall and Jones, in Am. Jour. Med. Sci., April, 1916. Also papers by Randall and by Lewis Fisher, Penna. Med. Jour., May, 1918, pp. 491, 492.

[2] Trans. Am. Laryng., Rhinolog., and Otolog. Soc., 1916.

[3] Jour. Am. Med. Ass'n, vol. lxiii, pp. 1813-1818.

true ataxia but rather involuntary irregular movements, and an absence of the characteristic speech. Optic atrophy is not seen. The history and evolution are also different, chorea having a much more abrupt onset and a more acute course, with a tendency to recover. In insular sclerosis there is intention tremor and spastic gait, but the nystagmus and affections of speech may cause some resemblance, and the distinction should be made with care.

XX. DISEASES OF THE PONS.

The pons is well named, for it is a bridge by way of which many nerve-tracts take their course. It is also the seat of the nuclei of several important cranial nerves, namely, the fifth, sixth and seventh. The pyramidal or motor tracts from the brain pass down through the anterior parts of the pons, and the great sensory tract, known as the median and lateral fillet, passes upward through the deeper portion. The transverse fibres of the pons connect the hemispheres of the cerebellum with each other, and other important cerebellar connections are probably made through the middle peduncles; and finally the cochlear nerve, or nerve of hearing, enters the lower outer part of the pons, and it, as well as the auditory tract to the posterior quadrigeminal bodies, passes through the mid-region of this great bridge.

Pathology.—Tumors, hemorrhage, softening and meningitis are the chief lesions here, as in other regions of the brain. A syphilitic infection of the membranes and basilar artery may cause softening, or may involve the roots of the nerves on the surface of the pons.

Symptoms.—In such a complicated structure the symptoms vary widely; one of the most characteristic symptoms is the hemiplegia alternans, in which there is an opposite hemiplegia with paralysis of the sixth nerve causing internal strabismus, and of the seventh nerve causing facial paralysis on the side of the lesion; in some cases the fifth is also involved, also the eighth. The sixth nerves, which pass out near the median line, may both be involved even in a lesion which is mainly unilateral. The above symptom-complex points to a lesion in the lower and anterior half of the pons, and if it is a tumor it is most likely to be meningeal and located in the cerebellopontile angle. Cushing has sought to establish a syndrome for tumors of the acoustic nerve in the cerebellopontile angle, in which an affection of the cochlear, or auditory part proper, of this nerve is an early or initial symptom.[1] Mills has reported a case of limited softening in the lower ventral part of the pons near the median line, in which paralysis of the left sixth and paresis of the right sixth nerve were associated with left hemiplegia. There apparently was no facial palsy. The facial nerve may exhibit remarkable resisting power, as in a case observed by Lloyd, in which the seventh nerve was bent over the surface of a tumor in this region, and yet there had been no paralysis of the face. In some cases both roots of the auditory nerve are not simultaneously involved: as one of these roots subserves hearing (the cochlear nerve) and the other probably subserves equilibration (the vestibular nerve), it is well to test these two functions separately.

[1] Tumors of the Nervus Acusticus.

If the lesion is high in the pons, above the level where the motor tracts for the sixth and seventh nerves decussate, the paralysis of these nerves will be on the side opposite the lesion and on the same side as the hemiplegia.

Involvement of the fifth nerve causes anæsthesia of one side of the brow, face, and tongue (in whole or in part, according to the extent of the involvement), the eyeball of the affected side, and paralysis of the muscles of mastication—the temporal, masseter, and pterygoids. A neuroparalytic ophthalmia may result and totally destroy the eye. An early symptom may be an abolition of the corneal reflex.

Superficial or meningeal lesions, unless they make deep pressure, are not likely to involve the sensory tract (the fillet), which lies deeply within; nevertheless in all cases of suspected pontile lesion the limbs should be carefully tested for anæsthesia, including the tactile, the thermal, and the pain senses.

Deep lesions of the pons may cause headache, vertigo, hemiplegia, hemianæsthesia, dysarthria, paralysis of the tongue (not nuclear), convergent strabismus or even lateral deviation of the eyes, inability to swallow, and intense emotionalism, with involuntary spasmodic laughter in some cases. Rotation of the head to one side has been noted; also profuse sweating, vasomotor symptoms, and even epistaxis. Conjugate deviation of the head and eyes is said to be caused sometimes by a lesion high in the pons. Deviation of the eyes is *away* from the side of the lesion if this is in the posterior longitudinal fasciculus; and cases have been reported in which the eyes were rolled to one side and the head to the other in the hemiplegia alternans.

The symptom-complex described as pseudobulbar palsy is probably caused in some cases by a pontile lesion.

The Bárány tests should be made in suspected cases of pontile lesions. (See page 740.)

Diagnosis.—This is made from the peculiar grouping of symptoms as given above. A question may arise as to the nature of the lesion, whether it be a tumor, a hemorrhage, a softening, or a syphilitic meningitis. Tumor is usually slow in onset and gradual in its course; hemorrhage and softening abrupt in onset and not progressive; syphilitic meningitis may not be easily distinguishable from tumor, but irregularity in the onset and course of the symptoms points to specific disease. The Wassermann and other laboratory tests should be made.

XXI. BULBAR PALSY.

In the account of progressive muscular atrophy and amyotrophic lateral sclerosis the degeneration of the ganglion cells in the anterior, or motor, horns of the spinal cord, which is characteristic of these diseases, is described. We have now to describe a disease which depends upon a similar degeneration of motor ganglion cells, but these cells are located higher in the medullary gray matter and preside over special functions; they are the motor neurons which arise in the bulb, or medulla oblongata, and especially in the nuclei of the ninth (glossopharyngeal), tenth (pneumogastric), and twelfth (hypoglossal) nerves. This disease is known as bulbar palsy, or labio-glosso-pharyngeal paralysis.

FIG. 382.—Woman with deviation of the eyes toward the right, of the head toward the left. Case of hemiplegia alternans inferioris encephalitidis pontis (crossed hemiplegia from inferior encephalitis of the pons).—Oppenheim.

FIG. 383.—Atrophy of tongue due to partial bulbar paralysis.—Lloyd.

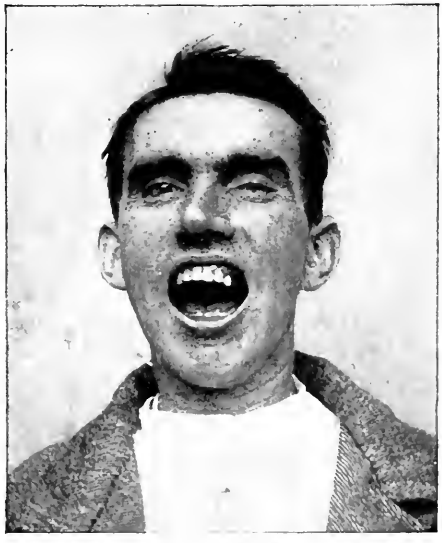


FIG. 384.—Pseudobulbar palsy, showing paralysis of the lips, tongue, and lower jaw, from lesions in the lenticular nuclei.—Lloyd.

Pathology.—There is found a degeneration of the large multipolar cells in the nuclei of origin of the ninth, tenth, and twelfth nerves in the medulla, and possibly of the seventh nerve in the pons. Thus in the nucleus ambiguus, which contains the motor cell-bodies of the ninth nerve, these multipolar cells are found greatly changed; they have shrunken in size, present evidence of chromatolysis and displacement of the nuclei, and the nerve-fibrils are diminished in number. In advanced or severe cases it is evident that many cell-bodies have entirely disappeared. The same changes are found in the nucleus of the twelfth nerve, which is entirely a motor nerve. In the case of the ninth, which is a mixed nerve, the sensory ganglia (the jugular and petrous) are not involved. The diseased cells, however, are not confined to the regions just mentioned, but are found in that rather extensive mass of gray matter in the bulb from which arise motor fibres not only for the ninth but also for the tenth (pneumogastric) and even the spinal accessory. In some cases the roots of the bulbar nerves are degenerated, and occasionally some degeneration is observed in the pyramidal tracts of the cord. This sclerosis of the motor columns marks the connection of this disease with progressive muscular atrophy and amyotrophic lateral sclerosis, for that there is some relationship is evident not only from the similarity of the degeneration in the motor nuclei, but from the fact that bulbar palsy may precede or complicate either of these two diseases, especially the latter.

Symptoms.—The initial symptoms usually are disorders of speech. The articulation becomes imperfect, especially for labials and linguals, due to beginning paresis of the lips and tongue. Nasal speech occurs, and finally a very distressing dysarthria, in which the patient finds it almost impossible to make himself understood. The attempt at speech is fatiguing, and finally may be almost abandoned. Deglutition in turn becomes impaired. The patient can manage the bolus of food only with difficulty, especially in passing it back into the pharynx. There may be regurgitation of fluids though the nose, or out between the paralyzed lips, and attacks of strangling, coughing, and vomiting result. Mastication also is somewhat impaired. Phonation is altered, and becomes monotonous. Respiration may also be embarrassed. The loss of power in the lips causes inability to whistle or to show the teeth. The lower part of the face becomes immobile and expressionless; the lips are flaccid and partially open, and drooling or dribbling of saliva results. The tongue becomes so palsied that it cannot be protruded, but lies almost or quite motionless in the mouth. The paralysis is atrophic; hence all the affected muscles waste and lose tone. The tongue is flabby, wasted, and fissured. The lips are thin and lifeless. The pharyngeal reflex may be abolished, but sensation is not involved. The velum palati hangs flaccid, and the laryngoscope may reveal paralysis of the adductors of the vocal cords. As a rule, the muscles of the upper part of the face and of the eyes are not involved.

Occasionally there is evidence of lateral sclerosis, as exaggerated knee-jerks and some spasticity of the gait. This indicates, as already said, the kinship of this disease to amyotrophic lateral sclerosis.

The electrical reactions may be partially altered, but true reactions of degeneration are seldom seen. This is for the same reason that holds

in progressive muscular atrophy; as long as any muscle fibres remain in connection with the gradually wasting nuclei they react to the current.

The onset of bulbar paralysis is usually insidious, the course is slow and chronic, and the disease is incurable. Acute cases, with rapidly developing symptoms, have been reported, but they are rare.

Diagnosis.—There is not much possibility of confusing true bulbar paralysis with any other disease. Diphtheritic paralysis has been mistaken for it, but in that disease there is no paralysis of the tongue and lips, and the history and course are different. The mistake is most likely to occur from regarding the paralysis of the velum palati and the dysphagia as evidences of an acute onset of bulbar paralysis; but the history of sore throat, the paralysis of accommodation, and the evidences of a multiple neuritis are usually sufficient to identify postdiphtheritic paralysis.

The distinction between true bulbar paralysis and myasthenia gravis presents some difficulty, but the subject is discussed in connection with the latter disease.

Organic disease, such as tumor of the pons or medulla, may simulate bulbar paralysis—may, in fact, cause a bulbar palsy—but other evidences will be present of gross organic disease, such as are described under the head of Tumors of the Brain.

There is an apoplectic bulbar palsy due to hemorrhage or vascular disease in the bulb and pons. It may simulate atrophic bulbar paralysis, but its sudden onset, often with apoplectiform symptoms, is characteristic. In some non-fatal cases there may even be a tendency for some of the earlier symptoms to improve.

An acute disease of the gray matter of the bulb, analogous to the acute anterior poliomyelitis of children, has been reported by Wernicke and others —the so-called *acute polioencephalitis inferior*. The history and nature of the attack are usually sufficient to distinguish it. The symptoms are those of bulbar palsy of rapid onset. Wernicke associates this disease with a similar affection of the nuclei in the mid-brain, causing ophthalmoplegia—the *acute polioencephalitis superior*. Syphilis is doubtless the cause of some of these abrupt or acute bulbar palsies.

XXII. PSEUDOBULBAR PALSY.

The term *pseudobulbar palsy* applies to a labio-glosso-pharyngeal paralysis which is of cerebral, not of nuclear, origin. To understand it we must bear in mind that there are centres in the motor cortex of the brain for the lips, the tongue, the muscles of mastication, the pharynx, and the larynx, and that these centres are connected with the nuclei of the facial, the motor branch of the fifth, the hypoglossal, the pneumogastric, and the glossopharyngeal nerves by the motor conducting paths, which run down through the internal capsule, cerebral peduncle, and pons. These nuclei are located in the pons and medulla oblongata. Hence a lesion which interrupts these motor tracts from the brain causes bulbar or pontobulbar symptoms: there is paralysis of the lips, tongue, and the muscles of mastication, of deglutition, and possibly of phonation.

Symptoms.—A few such cases have been reported, and the accompanying illustrations represent two such patients from the service of J. Hendrie

Lloyd in the Philadelphia Hospital. The nature and location of the lesions in such cases are not always clear. As the symptoms are usually bilateral, it is not easy to interpret them as due to a unilateral lesion. Sometimes the symptoms in their entirety occur only after sudden apoplectiform attacks, and the inference is that bilateral vascular lesions, such as could be caused by atheroma or syphilis, are the cause. In one of Lloyd's cases bilateral lesions were found in the lenticular nuclei. There may be hemiplegic or diplegic symptoms, not always well marked.

FIG. 385.—Pseudobulbar palsy; involuntary laughter.—Lloyd.

The muscles of the tongue and lips and of mastication and deglutition may be completely paralyzed. In one of the cases here depicted the tongue was motionless, the mouth hung open because of paralysis of the temporal and masseter muscles, the lips were paralyzed, and the patient could only swallow by thrusting the bolus of food far back into his pharynx with his finger.

The paralysis is central, as shown by the absence of muscular atrophy, of fibrillation, and of the reactions of degeneration. A peculiar symptom is spasmodic involuntary laughter or crying. It is well shown in the cuts. Brissaud thought that this indicated lesions of the optic thalami.

FIG. 386.—Involuntary laughter in a case of pseudobulbar palsy.—Lloyd.

Cerebral symptoms sometimes occur in these patients, such as aphasia, dysarthria, dementia, hemianopsia, etc. There is also seen in rare cases conjugate paralysis of the eyes; in one of the above cases there was paralysis of the upward movement of both eyes.

Among other symptoms rarely seen are optic neuritis or atrophy, and respiratory troubles. Anæsthesia is not commonly observed.

There has been more speculation than actual post-mortem observation about the seat of the lesion; and Oppenheim, who reviews the subject, comes to no very definite conclusion. Brissaud places the lesion, or lesions, in the posterior part of the optic thalami;[1] this was not so in Lloyd's recent case.

[1] Leçons sur les Maladies Nerveuses, Paris, 1895, p. 446.

Diagnosis.—The disease is distinguished from true bulbar palsy by the abrupt onset, the central character of the symptoms,—the absence of atrophy, of fibrillation, and of electrical changes,—by the associated cerebral symptoms, and by the history.

DISEASES OF THE CRANIAL NERVES.[1]

The cranial or cerebral nerves comprise twelve pairs of symmetrically arranged nerve-trunks which are immediately connected with the brain and pass through various foramina at the base of the skull to be distributed, with the exception of the tenth pair, to the structures of the head and neck.

These pairs of nerves are numbered according to the order in which they penetrate the dura from before backward from the first to the twelfth. They have, moreover, received designations descriptive of their functions or distribution. Some of them are wholly motor; others convey impulses of special sense; while certain of them transmit impulses of common sensation and motion.

THE CRANIAL NERVES.

No.	Name.	Function.
I	Olfactory	Special sense of smell.
II	Optic	Special sense of sight.
III	Oculomotor	Motor to eye muscles and levator palpebræ superioris.
IV	Trochlear	Motor to superior oblique muscle.
V	Trigeminal	Common sensation to structures of head. Motor to muscles of mastication.
VI	Abducent	Motor to external rectus muscle.
VII	Facial	Motor to muscles of head (scalp and face) and neck (platysma). Probably secretory to submaxillary and sublingual glands. Sensory (taste) to anterior two-thirds of tongue.
VIII	Auditory, (a) Cochlear division (b) Vestibular division.	Hearing. Equilibration.
IX	Glossopharyngeal	Special sense of taste. Common sensation to part of tongue and to pharynx and middle ear. Motor to some muscles of pharynx.
X	Pneumogastric or vagus	Common sensation to part of tongue, pharynx, œsophagus, stomach, and respiratory organs. Motor (in conjunction with bulbar part of spinal accessory) to muscles of pharynx, œsophagus, stomach and intestine, and respiratory organs; inhibitory impulses to heart.
XI	Spinal accessory	Spinal part: Motor to sternomastoid and trapezius muscles.
XII	Hypoglossal	Motor to muscles of tongue.

Modern knowledge concerning the relative position of the cell-bodies of motor and sensory neurons renders necessary a readjustment of the former views concerning the superficial and deep origin of the cranial nerves and their course from the brain to parts outside the skull. Only the motor fibres of the cranial nerves arise from nerve-cells within the cerebrospinal axis, while the fibres which transmit sensory impulses have their origin from cell-bodies forming ganglia situated outside of the central nervous system and in the course of the nerve-trunks. The term "deep origin" as indicating cell-groups constituting nuclei within the brain and "superficial origin" as indicating the point of attachment to the surface of the brain can only be properly employed in regard to motor nerves and the fibres of motor and sensory nerves which convey motor impulses.

[1] This section on Diseases of the Cranial Nerves was written by the late Dr. William Pickett.

The cell-groups with which the terminal arborizations of the sensory fibres come into relation within the cerebral substance are not nuclei of origin but of termination—nuclei of reception. The impulses which they receive are transmitted to various parts of the brain by neurons of the second, third, or even higher order. The motor nerves then have their deep origin within the substance of the brain, their superficial origin at the point of their attachment to the surface of the brain, and their exit from the skull by the various foramina. The sensory nerves have their origin in their respective ganglia, their entrance into the skull by way of certain foramina, their points of attachment to the brain, and their nuclei of reception. Finally, the nerves of common sensation and motion, viewed from the standpoint of the direction of the impulses which they convey, whether they be afferent or efferent, contain fibres which enter the brain and fibres which make their exit by way of the respective foramina.

Every cranial nerve is directly or indirectly in relation with groups of neurons in the cerebral cortex. These groups constitute the higher cortical centres, the location of which in the case of many of the nerves has been more or less accurately determined.

I. FIRST NERVE.

The term olfactory nerve, formerly employed to designate the olfactory bulb and tract as well as the filaments, is now employed to describe the paths of conduction represented by a number of minute filaments which connect the perceptive elements situated within the Schneiderian mucous membrane with the olfactory lobe. In man the olfactory bulb and tract with its roots represent as rudimentary structures the more developed olfactory lobe of animals in which the sense of smell is keen. The true olfactory nerves, which number about twenty, are the axons of the neurons—the olfactory cells—which are situated in the olfactory area. This space is limited in extent, comprising on the outer nasal wall less than the mesial surface of the superior turbinate bone and a slightly more extended distribution upon the upper part of the nasal septum. These filaments pass upward by way of openings in the cribriform plate of the ethmoid bone and enter the olfactory bulb by its under surface.

Lesions of the nasal mucous membrane involving the olfactory area or the upper turbinate bone or the adjacent part of the septum, are attended with impairment or loss of the sense of smell. Lesions of the uncinate gyrus may also cause loss of smell upon one or both sides. The conduction path may also be destroyed in fractures of the base of the skull in the anterior fossa, involving the cribriform plate. Irritative lesions cause perversion of the sense of smell—parosmia; destructive lesions partial or complete loss—anosmia. Hallucinations of the olfactory sense may be symptomatic of hysteria, insanity, or tabes, and constitute one of the various forms of aura in epilepsy.

The sense of smell may be tested by presenting to each nostril in turn bottles containing familiar aromatic substances, as the oils of clove, peppermint, or asafœtida.

II. SECOND NERVE.

The ganglion cells among the rods and cones of the retina are the beginnings of the optic nerve; its apparent origin at the papilla is simply the point where the axons from these retinal cell-bodies, coming together, form the trunk of the nerve.

The ophthalmoscope is of special value in neurologic diagnosis because it lays bare, in the papilla, a great nerve close to the brain. Of the lesions thus directly revealed, papillitis or optic neuritis is usually a symptom of intracranial pressure or inflammation. When attended with much swelling it becomes "choked disk," since the optic foramen, unyielding, squeezes the swollen fibres. For the same reason optic neuritis is prone to pass into optic atrophy; but the latter is often primary, as in tabes, the optic nerve being, like other sensory roots, liable to degeneration in this disease.

For the consideration of vision in neurology, the retina is divided into lateral halves. The fibres from the right half-retinas (nasal half of left retina, temporal half of right) run together at the chiasm to form the right optic tract. Their arrangement is like that of the lines for driving a team of horses, the right line (as the optic tract) dividing to go to the right side of each horse's head (as to the right half of each retina) and, *mutatis mutandis*, the same applies to the left half-retinas. The partial decussation at the chiasm, then, is a device to make the two eyes, like a team of horses, act as one (binocular vision).

The optic tract passing back winds around the brain-stem (crus) to enter it dorsally after the manner of spinal sensory roots. It meets here its superior cell-bodies in three structures, the pregeminum, the pregeniculum, and the pulvinar, which constitute the "primary optic centres." From these cell-bodies, axons (the optic radiations) arise to pass into the posterior part of the internal capsule and outside the posterior horn of the lateral ventricle to the cortical optic centre in the cuneus, or, more exactly, in the region bordering the calcarine fissure. Lesion at any point in this path from the chiasm to the occipital cortex affects the half-retinas of the same side. Thus it appears that physiologically there is no cross-way in the optic path within the brain; yet just as an object touching the left side of the body is felt in the right half of the cerebrum through a crossing within the brain-stem, so an object on the left (in the left half-field of vision) is *seen* by the right half of the cerebrum through a *crossing*, not of nerve-fibres but of *rays of light within the eyeballs* (vitreous chambers); and as lesion of the right touch-path (fillet) causes left hemianæsthesia, so lesion of the right optic path, by affecting the right half-retinas, causes blindness of the left half-fields, called left hemianopsia. The varieties of hemianopsia are named by the fields which are darkened, not by the blind part of the retina. Tumor of the pituitary body by pressure at the chiasm destroying the inner fibres of each nerve from the nasal half of each retina causes bitemporal hemianopsia. More severe pressure may cause total blindness.

The optic tract may be pressed upon by tumor at the base, sometimes growing from the temporal lobe. There is hemianopsia, with Wernicke's sign and general symptoms of brain tumor. The primary optic centres may be the seat of a tumor or they may be pressed upon by a tumor of the middle lobe (vermis) of the cerebellum.

The optic radiations may be involved in tumor, hemorrhage, or softening at the hind part of the internal capsule, adding hemianopsia to the symptoms of capsular lesion; or one of these lesions may implicate the radiations farther back in the subcortex, when hemianopsia may exist alone; or a lesion in the angular gyrus may invade the radiations beneath, adding hemianopsia to the cortical symptoms of mind-blindness, etc.

Lesions in the vicinity of the calcarine fissure cause hemianopsia; occasionally they are bilateral, causing double hemianopsia which amounts to total blindness (amaurosis). The half-retinas are, as it were, mapped out upon the cuneus, so that, half of it being destroyed, there is blindness in a quarter of the opposite fields (quadrantic hemianopsia). Color appears to be separately represented. On the outer surface of the cerebrum, in the angular gyrus, apparently, is a higher centre for visual concepts. With lesion there the patient has mind-blindness, including word-blindness.

The mid-brain, receiving the great sensory eye-nerve (optic), sends back to the eye its chief motor nerve (third or motor oculi). These two nerves are the limbs of the reflex-arc through which the pupils react to light; and lesion of either may cause among other symptoms impairment of the light reflex.

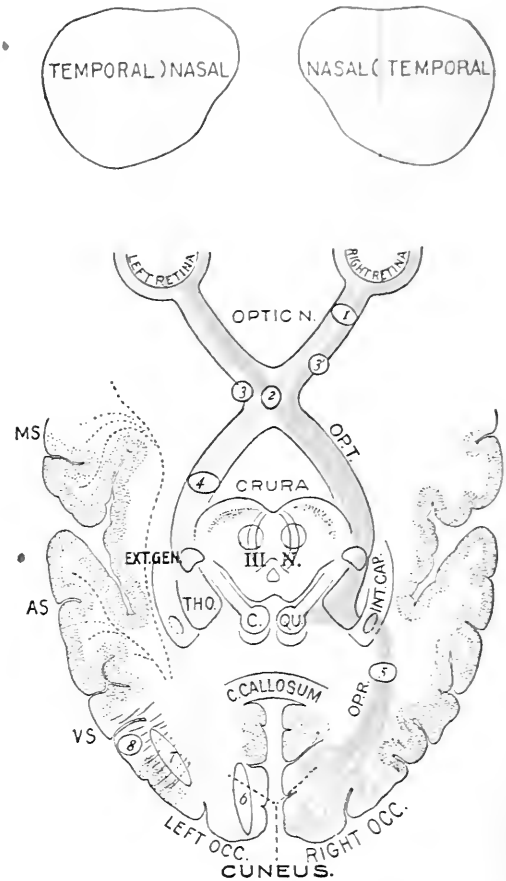


Fig. 387.—Diagram of visual system. Modified from Vialet. OP. T., optic tract; INT. CAP., internal capsule; OP. R., optic radiation; THO., optic thalamus; EXT. GEN., external geniculate body; C. QU., corpora quadrigemina; MS., motor speech centre; AS. auditory speech centre; VS., visual speech centre.

Lesions at the points indicated by the figures in the diagram cause the following morbid conditions: 1, blindness of the corresponding eye; 2, bitemporal hemianopsia; 3, nasal hemianopsia; 3, and 3′, binasal hemianopsia; 4, right lateral homonymous hemianopsia with Wernicke's hemianopic pupillary inaction sign; 5, left lateral homonymous hemianopsia with normal pupillary reflexes; 6, right lateral homonymous hemianopsia with normal pupillary reflexes; 7, amblyopia (especially on the side opposite the lesion); 8, on the left side, word-blindness.

Wernicke's "hemianopic pupillary inaction" is a sign of lesion at the base of the brain (see p. 373).

The third nerve nuclei just beneath the anterior gemina are connected with these bodies, or with the optic tract in front of them, by collateral fibres to complete the light reflex arc, and lesion (usually tabetic or paretic degeneration) of these collaterals may impair the light reflex alone, causing

reflex iridoplegia without other eye-symptoms. Loss of the light-reaction of the iris with the preservation of the reaction in convergence and accommodation is called the Argyll-Robertson pupil. It occurs in tabes and in paresis. But abolition of the reflex to light is also seen in cerebrospinal syphilis: as is also the condition known as internal ophthalmoplegia, or abolition both of the light reflex and of the movement on accommodation.

OPTIC NEURITIS—PAPILLITIS.

Inflammation of the optic nerve, visible with the ophthalmoscope. When the swelling causes bulging of the nerve-head to the extent of two diopters or more it is called choked disk. An affection of the nerve back of the eyeball, causing peculiar symptoms, is called retrobulbar neuritis. Sclerosis of the nerve, seen in the papilla, is optic atrophy. Ordinarily, as the sequel of optic neuritis, it is consecutive atrophy; when a part of the degeneration in tabes, etc., it is primary; when a symptom of brain disease, like tumor, secondary optic atrophy.

Impairment of vision and of the iris-reflex to light, both in varying degree, with ophthalmoscopic changes in the disk, indicate disease of the optic nerve. The field of vision is contracted, sometimes irregularly. The diagnosis rests mainly upon ophthalmoscopic examination; the prognosis upon the cause of the condition. In general it is grave.

III. THIRD, FOURTH, AND SIXTH NERVES (MOTOR NERVES OF THE EYE).

Supplying the internal muscles of the eyeball, except the dilator fibres of the iris, and the external muscles, except the external rectus and the superior oblique, the third nerve is the most important motor nerve of the eye, whence its name, *motor oculi*.

As cortical centres control movements, not muscles or nerves, the third nerve with its opposite actions is not totally affected in cerebral palsies. When in a case of head injury one pupil is dilated and immobile, this (Hutchinson pupil) is said to be pathognomonic of extradural hemorrhage.

The internal rectus, supplied by the third nerve, when paralyzed, permits the eyeball to turn outward (divergent strabismus); there is double vision with the secondary image on the opposite side (crossed diplopia). The inferior rectus being paralyzed, the eyeball fails to move downward and to some extent outward; of the double vision, the secondary image is below. The superior rectus paralyzed, the eyeball does not move upward, nor perfectly outward; the secondary image is above. To look with this eye the head is thrown back. The inferior oblique is opposite, in action and in the effects of paralysis, to the inferior rectus. The superior oblique, supplied by a separate nerve, the fourth or trochlear, is often paralyzed; the effects are opposite to those of paralysis of the superior rectus. The fourth nucleus, under the posterior geminum, is a continuation of the third. The sixth nucleus, in the lower part of the pons, is another link in the chain of gray matter for the ocular muscles. The external rectus, supplied by the sixth or abducent nerve, is more often affected alone than any other ocular muscle. When the entire third nerve is paralyzed the eyelid droops (ptosis), the eye turns outward by the action of the sixth nerve, and slightly downward by the action of the fourth, the

pupil is larger than its fellow and fails to react to light or in accommodation. Lesions of the trunk of the nerve are generally unilateral; they may affect the extra-ocular muscles, while sparing the iris-movements and accommodation. The third nerve-trunk in the orbit may be injured, as by a blow on the temple, or compressed by an orbital growth. It may be the seat of neuritis, from rheumatism, alcoholism, or diphtheria, or of degeneration in tabes. Within the skull it may be implicated in meningitis or compressed by tumor or aneurism. In its course through the crus it may be compressed by tumor, or suddenly paralyzed by hemorrhage, embolism, or thrombosis, commonly associated, by implication of the motor pathway, with hemiplegia of the opposite side (Weber's syndrome). In the cortex there is no representation of the third nerve as a whole, but of the various movements governed by it. In traumatism of the convexity on one side inducing extradural hemorrhage, the pupil of this side may be dilated and immobile (Hutchinson pupil). Lesions, as apoplexy, affecting the motor pathway within the cerebrum often cause conjugate deviation of the eyes, with the head ordinarily toward the side of the lesion. Finally, syphilitic disease, either gumma, meningitis, or neuritis, often selects the third nerve. The palsy of myasthenia gravis is often in the domain of the third nerve (recurring palsy—ophthalmic migraine).

Nuclear Ocular Palsies.—(See p. 719.)

IV. FIFTH NERVE.

The fifth or trifacial is the great nerve of common sensation for the head. Its motor branch, for mastication, is subsidiary. The surfaces sup-

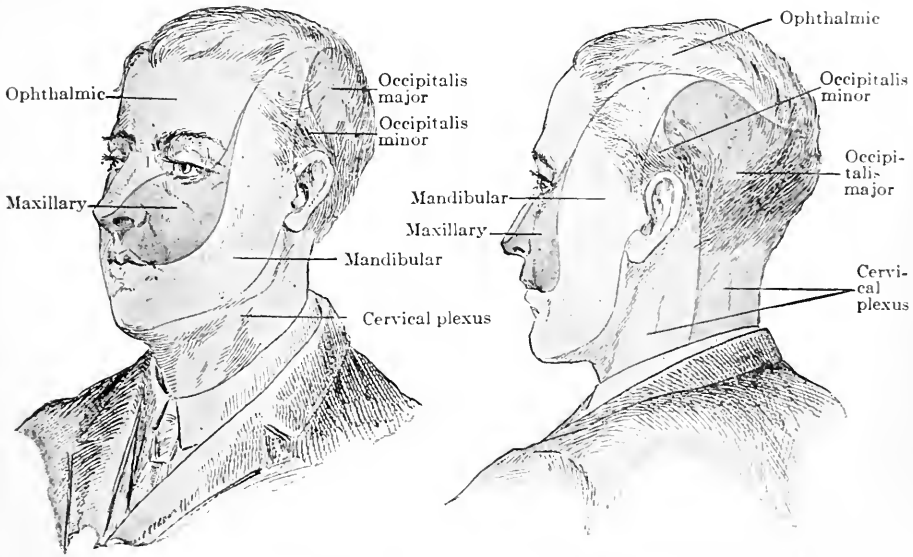


FIG. 388.—Showing distribution of cutaneous branches of trigeminal and cervical spinal nerves.—Piersol.

plied by its three branches, namely, the ophthalmic and the superior and inferior maxillary nerves, are shown in the accompanying illustrations.

Entering the cranium—the first branch by the sphenoidal fissure, the second by the foramen ovale, the third by the foramen rotundum—the branches unite in the Gasserian ganglion, thence to enter the side of the pons, midway between its upper and lower borders. At this level, in the back of the pons is the main nucleus of the fifth, but a chain of gray matter and connecting fibres (mid-brain root) extending alongside the aqueduct of Sylvius forms the motor root, which leaves the pons just above the sensory root and passes under the Gasserian ganglion, and a similar chain descending at the side of the medulla conveys sensory impulses down to the cervical cord.

Diseases of the Fifth Cranial Nerve.—Branches of the fifth may be the seat of neuralgia, from cold or from dental affections; they may be damaged by wounds. Sclerosis of the Gasserian ganglion may be the cause of facial hemiatrophy; it is the usual cause of trifacial neuralgia or tic douloureux, and may be the seat of irritation, giving rise to herpes zoster of the face. Hemorrhage, tumors, or other lesions within the pons, paralyzing the fifth, cause anæsthesia of various areas of the face. Meningitis, syphilitic lesions, tumors beneath the pons often implicate the roots of the fifth.

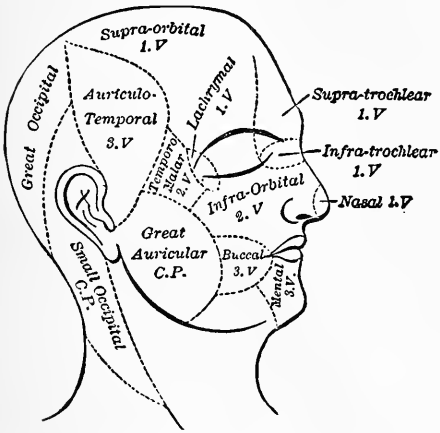


FIG. 389.—Normal distribution of the fifth nerve to the face. *1. V*, ophthalmic division; *2. V*, superior maxillary; *3. V*, inferior maxillary. The names on the different areas indicate the branches supplying them. (Flower.)—Posey and Spiller.

Symptoms.—Disease of the fifth nerve may cause first neuralgic pain, but the chief effect is anæsthesia in the distribution of one or more of its branches to the middle line of the face. A touch upon the conjunctiva is not felt, and does not excite the flow of tears. Fumes in the nostril have no effect, and on the tongue, especially its anterior two-thirds, substances whose taste-qualities are allied to touch are not recognized. The salivary secretion fails, the mucous membranes are dry, and from slight injury ulcers may form upon them, particularly over the cornea, which becomes clouded, opaque, and may perforate, leading to panophthalmia (neuroparalytic ophthalmia).

The motor portion of the fifth being paralyzed, the jaws are not closed so firmly on the affected side, and in opening deviate toward that side. The weaker action of the temporal and masseter can be felt by the fingers.

Diagnosis.—Anæsthesia of half the face, including the mucous membranes, when it exists alone points to lesion of the Gasserian ganglion or of the nerve-trunk between this and the pons. Anæsthesia corresponding to one branch of the fifth may be due to lesion at any point in the course of the branch. When the fifth nerve and the cranial nerves next in order—the sixth or seventh, fourth, or third—are affected together, the lesion is at the base of the brain involving the roots of these nerves. Anæsthesia of one side of the face and of the arm and leg of the same side (hemian-

æsthesia) points to lesion in the posterior third of the posterior limb of the internal capsule; but of the face on one side and of the arm and leg opposite (crossed anæsthesia) lesion in the pons, on the side of the facial anæsthesia. With the latter there may be loss of the associated movement of the eyes to this side and there may be a corresponding crossed motor paralysis.

Differential Diagnosis.—Hysterical hemianæsthesia is not associated with dryness of the mucous membranes; the tears flow on irritation of the conjunctiva and the special senses are affected on the anæsthetic side.

Tic Douloureux.—This is an aggravated and persistent form of neuralgia in the trigeminal nerve.

Pathology.—The disease has often been described as idiopathic, but recent observations, especially by Horsley, Rose, Putnam, Spiller, and others, have tended to show that there are degenerative or sclerotic processes in the nerve-fibres and in the Gasserian ganglion. The tendency for the disease to pass slowly but surely from one branch to the other, even after the branch first affected has been excised, seems to indicate that the process spreads from one group of neuron cells to the others in the Gasserian ganglion in somewhat the same way as the motor neurons of the anterior horns of the spinal cord are involved in progressive muscular atrophy. The essential causes of this process are obscure.

Symptoms.—The chief and usually the only symptom is pain. Lachrymation, flushing of the face, and spasmodic movements of the facial muscles are occasionally seen. Herpes has been observed in some cases, but it is doubtful whether it belongs to the disease proper.

The pain is intense, atrocious, even agonizing. It sometimes occurs in paroxysms or exacerbations, but in many cases there is more or less constant suffering. The paroxysms are usually spontaneous, but they can also be excited by trifling causes, such as movements of the face, attempts at talking or eating, or even a draught of cold air. It is characteristic of tic douloureux to begin in one division, or even in one branch, of the fifth nerve, and then to spread in time to other branches. The progress is usually slow and chronic. Many cases are operated on, but excision of the offending branch, while often giving relief for longer or shorter periods, seldom effects a radical cure, the pain returning in another branch.

Spasm of the facial muscles is seen in some cases and even constitutes a special type of the disease (the so-called convulsive or epileptiform tic), but it is not common. The pains in these cases are usually paroxysmal and severe; they occur with lightning-like quickness, and the facial muscles are thrown into twitchings and spasmodic movements. The taking of food is sometimes seriously interfered with by the pain.

Paralysis and anæsthesia are not seen in tic douloureux. Inhibition of movement is caused by the pain and the fear of pain, but neither the facial nor the masticatory muscles (the latter of which are supplied by the motor branch of the fifth nerve) are truly paralyzed. Anæsthesia in the territory of the fifth nerve is also absent as an almost universal rule; a few exceptions have been noted, but they properly raise a question whether the case is typical. Neurotrophic disorder of the eye, as seen in organic disease of the fifth nerve, is also not observed. The affection is unilateral.

Diagnosis.—The disease is unmistakable. The gradual establishment of severe pain in one branch of the fifth nerve, its progress in time to other branches, its intractability, and the facial spasms (when they occur) are easily recognized. The only doubt that may arise is with reference to the causation and pathology.

Organic lesions of the fifth nerve, such as occur from tumors, meningitis, etc., may cause pain, but usually they also cause anæsthesia of the face, brow, eye, and tongue, and paralysis of the masticatory muscles, and the pain is not always intense or strictly limited to a branch of the trigeminus. Moreover, in such cases the symptoms are seldom confined to the fifth nerve.

The term "epileptiform," as applied to the type in which facial spasms occur, is a misnomer. The disease has no relation to epilepsy.

Masticating Spasm.—Tonic spasm in the domain of the motor fifth occurs as trismus or "lockjaw" in tetanus; occasionally in tetany, in hysteria, and reflexly in dental affections, like caries of a molar. Clonic spasm, noticeable in a chill and in the epileptic convulsion, occurs rarely as an isolated affection called "chattering teeth."

V. SEVENTH NERVE.

The seventh or facial nerve, arising from a nucleus in the pons, passes behind and over the sixth nucleus and out at the side of the pons near its lower border. With the eighth it enters the internal auditory meatus, then alone passes in the Fallopian canal close to the tympanum, and finally, through the stylomastoid foramen, emerges upon the face.

Paralysis of the facial muscles may be supranuclear or central as the result of lesion of the centre in the lower Rolandic cortex, or of the fibres from this centre passing down through the brain, commonly as a part of hemiplegia; or nuclear in consequence of lesion of the nucleus in the pons; or infranuclear from lesion of the nerve-trunk at any point. The ordinary form of facial palsy is "peripheral" from neuritis in the Fallopian canal, and is called Bell's palsy. Hemorrhage or softening in the pons, damaging one facial nucleus, may paralyze the face on that side and affect the adjacent motor pathway, the arm and leg of the other side (crossed paralysis). The seventh nerve may also be paralyzed in that rare form of tetanus known as cephalic tetanus.

At its emergence from the pons the seventh nerve may be implicated in meningitis, or compressed by a new growth, which may also involve the sixth and eighth nerves. Within the Fallopian canal the seventh nerve may be encroached upon by caries of the temporal bone from middle-ear disease. It may be damaged in operations.

Bell's Palsy.—This affection is ascribed to neuritis from exposure. The nerve swells in its bony case and is compressed. The corresponding half of the face is rapidly paralyzed.

Symptoms.—The lines of expression are smoothed out; the mouth droops on that side, and the lower eyelid sags and lets the tears run down. In "showing the teeth," the mouth and cheek are dragged toward the sound side; in looking up, the forehead does not wrinkle on the affected side; and in the attempt to close the eye, the lids remain apart.

The palate moves symmetrically and the tongue is protruded in the middle line, though by the distortion of the mouth it appears to deviate. Liquid, in drinking, or saliva runs from the corner of the mouth, and in chewing the food gathers in the cheek. The sense of taste on the front of the tongue, supplied by the chorda tympani, is impaired in some cases, as this nerve accompanies the facial within the Fallopian canal for a short distance. The reaction of degeneration occurs typically in facial palsy. Both nerve and muscle show diminishing irritability to faradism after a few days, while to galvanism the muscle contracts excessively, and in the serial order of the reaction of degeneration. Bell's palsy is rarely sudden, but usually rapid, developing in a few hours or days. It lasts ordinarily two or three months. In severe cases, after four or five months, contractures of the affected muscles deepen the lines of expression, so that the face appears normal or the sound side looks weaker.

Diagnosis.—In recent cases the condition is obvious. In older ones the muscular contracture and overaction may conceal it; but strong movements in showing the teeth or closing the eyes will show the difference of the two sides. Cerebral (supranuclear) paralysis of the facial is usually a part of hemiplegia. In this form the upper half of the face (orbicularis palpebræ, frontalis, and corrugator supercilii) regains power in a few days, through its bilateral innervation from the cortex; and even the lower half moves fairly with emotion, as in quiet smiling. In the cerebral form the supra-orbital reflex is preserved. Lesion at the base of the brain is indicated by concomitant paralysis of adjacent nerves, particularly the sixth and eighth. Deafness with facial palsy may result from tumor also involving the eighth nerve. In peripheral (nerve-trunk) palsies the entire half of the face is affected for all movements, voluntary or emotional, and the electrical reaction shows degeneration.

Prognosis.—Early return of power though slight is a good sign. Toward the end of the second week of paralysis an electrical examination gives valuable information. If at this time the faradic irritability is simply lessened, the paralysis will disappear in about two months; if lost, the outlook is bad, though some return of power is possible after several months. With the loss of faradic irritability occur the true reactions of degeneration to the galvanic current.

Facial Spasm.—As a symptom this occurs in epilepsy and chorea, in facial paralysis, in cerebral palsies as a part of athetosis, and as habit spasm. The habitual occurrence of spasm in one or several muscle-groups of the face is called *convulsive tic*. The orbicularis palpebræ and the zygomatics are its most frequent seat. Convulsive tic is a disease of later middle life (forty-five to sixty) more frequent in women. Prolonged anxiety is a factor; also, reflexly, a great variety of painful affections, as caries of a tooth.

Symptoms.—In the usual form of convulsive tic the eye is squeezed shut and the angle of the mouth drawn out and up momentarily at intervals. It is generally made worse by disturbing emotions. The spasm may be more extensive, involving other muscles of the face, mouth, neck, or arms, and especially in the platysma, which stands out on the side of the neck. In severe cases the spasm occurs in numerous quick jerks or frequently repeated contractions in the course of two or three minutes.

Convulsive tic, usually slight at first, increases gradually in the intensity and frequency of the spasm, and in the extent of the musculature involved. It is likely to continue indefinitely, but sometimes ceases after years. Intermissions of several months may happen.

Diagnosis.—Facial spasm is unmistakable. The spasm may be symptomatic of some gross disease. True convulsive tic is idiopathic. Sources of reflex irritation in the teeth, eyes, etc., must be investigated. Intracranial disease, causing facial spasm, may be tumor or other lesion of the face centre in the cortex, or of the root of the seventh nerve beneath the pons. From such a cause the affected muscles often will be found paretic, or will become paralyzed.

VI. EIGHTH NERVE.

The auditory nerve is physiologically two nerves—the cochlear for hearing, the vestibular for equilibration. From the distributions in the internal ear (the cochlea and the semicircular canals) the two parts, united as the eighth nerve, pass from the internal auditory meatus into the side of the pons. Here the two parts of the nerve, again separating, embrace the inferior cerebellar peduncle, the cochlear on its outer side, the vestibular on the inner, to connect with various nuclei in the pons and thence to seek different central goals. The cochlear fibres pass up in the lateral fillet, and by way of the postgeminum and postgeniculum reach the auditory centre in the first temporal convolution. The vestibular fibres pass to the middle lobe of the cerebellum.

The Bárány tests of the labyrinth and the vestibular nerve are now much relied on by some observers for determining the location of lesions in the pons, cerebellum, and cerebellopontile angle. These tests require special skill and pertain to the otologist. They depend upon interference with certain *normal* reactions of the vestibular nerve apparatus. These normal reactions are vertigo, nystagmus, past-pointing and falling, when the individual is rotated in a revolving chair, or when the external ear is douched with warm or cold water.[1]

Deafness.—Total deafness from birth or early childhood, depriving the child of speech, constitutes deaf-mutism. Acquired deafness frequently depends on disease of the labyrinth; but this is often secondary to middle-ear disease, particularly of the chronic catarrhal variety, or to meningitis by extension through one of the foramina. Basal fracture often enters the internal ear. The eighth nerve at its junction with the pons may be involved in meningitis, aneurism, or tumor, particularly fibroma of the nerve sheath. Degenerative disease, as tabes, may attack the eighth nerve. Pontine lesions rarely affect this nerve; but at the level of the posterior geminum, in the hinder part of the internal capsule or in the first temporal convolution, the auditory pathway may be damaged by tumor, hemorrhage, softening, etc., causing deafness of the opposite ear. Impaired hearing may be functional, as in hysteria.

Symptoms.—If no objective signs of obstruction of the external meatus or disease of the middle ear are present, deafness may be ascribed to con-

[1] See footnote references, page 724.

ditions which affect the reception of sound in the labyrinth or its conduction by the auditory nerve, or to lesions involving the central auditory tract. This is especially the case when the deafness is unilateral. When the sound of a tuning-fork held against the mastoid process—bone conduction—has ceased to be heard but is again perceived when the instrument is moved to a position opposite the external meatus—aërial conduction—labyrinthine disease may be suspected. When in unilateral deafness the sound of a tuning-fork in contact with the vertex at the middle line is perceived more distinctly on the side of the deaf ear, the fault of hearing is due to the conducting apparatus; when it is heard more distinctly or only in the sound ear, the deafness is caused by labyrinthine disease. In the latter condition there is an interval varying from one to several seconds between the time at which the patient ceases to hear the sound and the examiner ceases to feel the vibrations of the fork. There are no direct means by which deafness arising from lesion of the auditory nerve in its course can, in the absence of the signs of involvement of adjacent structures, be distinguished from that caused by disease of the auditory centres.

The locality of the eighth nerve-root, spoken of as the cerebellopontile angle, is a favorite seat of tumor (fibroma) which grows from the sheath of this nerve. This is recognized by its pressure-effects, paralysis of the facial and external rectus on the same side, deafness, vertigo, and incoordination, the latter partly of cerebellar origin. Deafness may be an early or initial symptom in those cases of tumor of the acoustic nerve, as Cushing has recently insisted on; and involvement of the fifth nerve may occur among the pressure, or neighborhood, symptoms, and be first shown by abolition of the conjunctival reflex. Deafness from a higher seat, the quadrigeminal region, or the internal capsule (posterior extremity) is usually associated with hemianopsia, and sometimes with other disturbances on the same side. Cortical deafness is likely to be of special character (word-deafness, etc.) related to aphasia. Sudden deafness indicates a vascular lesion, especially hemorrhage, most frequently in the internal ear. Syphilitic inflammation may involve the eighth nerve and the labyrinth. Hysterical deafness may be recognized by the associated symptoms.

Auditory Irritation. — Uncomfortable acuteness of hearing (hyperacusis) is ordinarily hysterical, though observed occasionally in facial palsy. Tinnitus aurium embraces simple subjective noises, as ringing, hissing, and roaring, referred either to the ear or to some part of the head. More elaborate sounds, as words seemingly spoken in the ears, in other parts of the body, or at a distance, are called auditory hallucinations. Tinnitus is a common symptom in the various diseases of the internal ear, as well as of the middle ear and external meatus. Tinnitus may arise especially in elderly persons, without definite cause. It is common in neurasthenia. In some cases it has a pulsating character, and is then referred to vasomotor disturbance in the internal ear. Head injuries, sudden loud noises, and, above all, the habitual subjection to noise (as in boiler-makers) dispose to it. Tinnitus is commonly associated with partial deafness, but may be accompanied by hyperacusis.

Diagnosis.—Irritation of the cortical centre (first and second temporal convolutions) is a cause of hallucinations of hearing, not of simple tinnitus.

Tinnitus due to irritation of the eighth nerve-trunk is known by the associated symptoms. Disease of the internal ear is the commonest cause.

Prognosis.—In a case of organic origin the prognosis is that of the primary disease. In functional disease, like neurasthenia, the symptom subsides as the patient improves. In some instances tinnitus is stubbornly persistent.

Ménière's Disease.—An affection characterized by noises in the ear, sudden attacks of vertigo with nausea and vomiting, and nervous deafness, which in many cases is progressive. The attacks are often apoplectiform, with momentary loss of consciousness.

This disease was first described by Ménière in 1861. The term should be restricted to the affection characterized by the complexus of symptoms about to be described.

Etiology.—Age plays an important part in the predisposition. The affection is very rare in early life. In a large proportion of the cases the attacks first show themselves between forty-five and fifty-five, but they may come on much later. Men suffer more frequently than women. Nothing is known of the exciting causes.

Symptoms.—The disease is paroxysmal, the attacks occurring at irregular intervals, and very often in series, several of which may take place in one day or on successive days. Such series or single attacks may be separated by intervals of weeks or even months. The attack begins suddenly with tinnitus aurium and subjective or objective vertigo of such intensity that the patient, in order to prevent himself from falling, is obliged immediately to catch some support or to sit or lie down. If loss of consciousness occurs it is momentary. Occasionally ocular symptoms accompany the attack. These consist of diplopia or nystagmus. Forced movements may occur, and in the intervals of frequent attacks there is an impairment of equilibrium, so that the patient walks with difficulty. The attack is usually of short duration. As the vertigo passes off the patient is pale, breaks into a profuse sweat, suffers from nausea, or there may be actual vomiting. As a rule, there is no disease of the middle ear. When it is present the association is accidental. The deafness, which is nervous, usually affects one ear only. It is progressive but not often complete. When deafness becomes complete the vertigo ceases, the end organs of the nerve being destroyed.

Three principal theories have been suggested to account for the phenomena of Ménière's disease: 1. That the symptoms are due to lesions of the labyrinth. There is progressive degeneration of the nerve or its end organs. 2. That the disease is a vasomotor neurosis of the vessels of the labyrinth. 3. That the primary trouble consists in an affection of the centres for hearing and equilibration. Of these the first is at present generally accepted.

Diagnosis.—The direct diagnosis of Ménière's disease rests upon the paroxysmal vertigo, the apoplectiform seizure, the occurrence of tinnitus, nausea, and vomiting, and the progressive nervous deafness. The differential diagnosis between the vertigo which is so prominent a symptom and other forms of vertigo depends upon the association of the foregoing symp-

toms, the paroxysmal nature of the attack, and the absence of other pathological states usually attended with vertigo.

Prognosis.—This is uncertain. A small proportion of the cases terminate, after a variable duration, in complete recovery, with total loss of hearing in the affected side. More commonly the disease proves persistent and intractable, and, with periods of exacerbation and improvement for which no explanation is to be found, continues throughout life. In rare instances the symptoms are so severe that the patients become bed-ridden.

VII. NINTH NERVE.

The ninth, tenth, and eleventh nuclei form a continuous chain of gray matter, and the nerves a continuous line of fibres springing from the side of the medulla, in the order of their numbering. The ninth or glossopharyngeal, mainly sensory, supplies the back of the tongue, the soft palate, tonsils, and adjacent pharynx, also the Eustachian tube and middle ear. The muscles of the upper pharynx are probably governed by the ninth.

Tumors or meningitis affect the ninth usually in company with other nerves. Swallowing is embarrassed by lesions of the nerve-trunk or, as in glosso-labio-laryngeal paralysis, by degeneration of its nucleus.

VIII. TENTH NERVE.

The tenth nerve, termed the pneumogastric or vagus, arises in the medulla by a line of nuclei and fibres continued downward from those of the ninth. It is the chief of the "bulbar" nerves in the variety and importance of its functions, supplying motor fibres to the muscles of the pharynx, œsophagus, stomach, and intestines, and to those of the larynx, trachea, and bronchi; sensory fibres to the dura mater, external ear, pharynx, œsophagus, stomach, larynx, trachea, bronchi, and the pericardium; and spinal fibres to the heart, liver, spleen, pancreas, kidneys, suprarenal bodies, and intestinal blood-vessels. The "respiratory centre" and "cardiac centre" are thus contained in the vagus nucleus, though for these vital functions, as for vasomotor regulation and for the movements of the stomach and intestines, the sympathetic acts in connection with this nerve.

The tenth nucleus may be implicated in softening, hemorrhage, or tumor of the medulla, usually with adjacent nuclei, inducing paralytic effects in combination known as "bulbar symptoms." Degeneration of the tenth nucleus in glosso-labio-laryngeal paralysis (chronic bulbar palsy) and its inflammatory destruction in acute bulbar palsy are responsible for the impaired phonation, difficult swallowing (mainly), and embarrassed cardiac and respiratory action. In cerebral disease, particularly bilateral softening in the neighborhood of the internal capsules, these symptoms are due to the destruction of the motor fibres destined to these nuclei (pseudo-bulbar paralysis). The root of the vagus may be the source of these symptoms in like combination from basilar meningitis, tumor, or aneurism of the vertebral artery.

In the neck the nerve-trunk accompanying the carotid, or lower down winding over the subclavian, may be compressed by an aneurism or tumor,

or damaged in operation. This nerve is involved in toxic or infectious neuritis more frequently than is ordinarily thought. The affection of the pneumogastric nucleus or trunk may be of a degree to induce irritative symptoms (slowness of the heart's action, spasm of the larynx, and vomiting), or paralytic symptoms (paralysis of the larynx, embarrassed respiration, and rapid pulse).

The inferior or recurrent laryngeal nerve, branching from the tenth at the base of the neck, winds around the great vessels—the aorta on the left, the subclavian on the right side—and ascends back of the trachea to the larynx, of which it supplies the most important muscles.

IX. ELEVENTH NERVE.

The accessory fibres of the spinal accessory join the vagus nerve, of which they form mainly the recurrent laryngeal branch. The spinal portion, composed of several motor roots of the cervical cord, forms part of the cervical plexus and supplies the sternomastoid and the upper portion of the trapezius muscle. Spasm of these muscles causes torticollis. Paralysis of the muscles supplied by the spinal accessory results from degeneration of the cervical gray matter in progressive muscular atrophy, from lesion of the trunk, in meningitis or brain tumor, and from wounds, tumors, vertebral disease, etc., in the neck. Paralysis of the sternomastoid alone may result from a wound of this muscle, severing the nerve-trunk within it. In paralysis of one spinal accessory, the head cannot be turned to the other side, the sternomastoid and the upper border of the trapezius are relaxed and in time wasted, and all movements about the shoulder, as raising the arm, are embarrassed. When this paralysis is bilateral the head falls backward or forward, according as the sternomastoid or the trapezius is more affected.

FIG. 390.—Torticollis (Jochimsthal).—Young.

Bilateral paralysis of the spinal accessory is conspicuous in meningitis, especially the tuberculous form of childhood, and in progressive muscular atrophy. Lesion of the nerve at the base of the brain, including the accessory part (laryngeal fibres), paralyzes the vocal cords, and is likely at the same time to implicate the hypoglossal or the glossopharyngeal and paralyze the tongue or the palate.

Torticollis or Wry-neck—Accessory Spasm.—True torticollis is a deviation of the head due to abnormal action of the muscles supplied by this nerve. It may be a fixed deformity—congenital wry-neck—or. due to spasm—spas-

modic wry-neck. In congenital torticollis there is atrophy of neck muscles, principally the sternomastoid, in consequence of prenatal poliomyelitis or of injury to the muscles during labor. Contraction of the sternomastoid tilts the head toward the affected side and at the same time rotates the face toward the opposite side. The muscle stands out rigid.

Spasmodic torticollis is of the nature of *facial tic*, and like it may be either tonic or clonic. The position of the head ordinarily is governed by the sternomastoid, but in some cases there is backward tilting in consequence of contraction of the trapezius. When the affection is bilateral the trapezii draw the head backward,—retrocollic spasm,—aided by both sternomastoids, and the frontales muscles in association raise the eyebrows. Spasmodic torticollis appears usually in middle life, is more frequent in women, and has been ascribed to a variety of causes. Its source in typical cases is probably cortical. It may be ushered in by pain and stiffness about the neck, but as a rule the spasm sets in gradually. It generally centres in the sternomastoid and may be confined to it, but the trapezius of the same side and the splenius of the other are commonly associated with the sternomastoid in spasm. The head is tilted sidewise and slightly backward and twisted to the other side, more frequently the left.

In bilateral (retrocollic) spasm the face is turned upward and the eyebrows raised synchronously. Ordinarily with the tonic variety of torticollis, as it becomes intense, clonic spasms are associated. The intensity varies, and intermissions are frequent. The affection may involve various muscles of the arm or face. The affected neck muscles hypertrophy in time.

The diagnosis is obvious. The rotation of the head to one side and its slight inclination to the other side, on which the sternomastoid muscle stands out prominently, especially when this position is emphasized by clonic jerkings, cannot be mistaken. So-called rheumatic torticollis, "stiff-neck," is marked by its acute appearance, often after exposure, with lameness and tenderness of the neck muscles. "False torticollis" is an unnatural position of the head from gross disease in the neck, most frequently of vertebræ, as Pott's disease and spondylitis deformans. In these conditions the sternomastoid is prominent on the side to which the head is turned. Hysterical torticollis occurs in younger persons with other signs of hysteria. True torticollis is more common in middle life.

Congenital torticollis may be relieved by operation. Spasmodic torticollis is chronic and intractable. Often after increasing for years it becomes stationary. There are cases in which remissions are frequent and intermissions occur, sometimes lasting many months. The disease is of no consequence beyond annoyance and embarrassment. Patients sometimes complain of fatigue or pain in the affected muscles.

X. TWELFTH NERVE.

The hypoglossal nerve, governing the muscles attached to the hyoid bone, controls the movements of the tongue. Within the cranium and in the upper part of the neck it is near the pneumogastric and spinal accessory nerves, with which it often is associated in disease, and the lips have some nuclear connection of movement with the tongue. In bulbar paralysis,

acute and chronic, the hypoglossal nuclei are a focus of the disease; and they occasionally are degenerated in tabes and paresis. In hemiplegia the cerebral—supranuclear—fibres for the tongue are commonly included in the lesion between the lower part of the motor cortex and the hypoglossal nucleus in the medulla. The roots in the medulla may be damaged by hemorrhage or by tumor, which usually implicates the main pathway to the opposite arm and leg; or the roots emerging from the medulla may be involved, often with the tenth and eleventh, in meningitis, syphilis, or tumor. In the upper part of the neck various gross diseases or wounds may injure the hypoglossal trunk with the spinal accessory.

Paralysis of the tongue without sensory disturbance is the effect of hypoglossal lesion. When this is bilateral the tongue is motionless. When paralyzed on one side the tongue, protruded, curves toward the affected side, speech is thick, and chewing is awkward. The affected half in time wastes, shows fibrillary tremors, and is puckered with transverse folds. When the lesion is nuclear, as in bulbar palsy, the lips share in the atrophy; when it is cerebral the tongue shows no trophic change. Associated particularly with double hemiplegia, often slight in degree, paralysis of the tongue, with other bulbar symptoms, constitutes "pseudobulbar paralysis."

Paralysis of the tongue from cerebral disease is commonly a part of hemiplegia. Nuclear palsy is generally bilateral and a part of "bulbar palsy," recognizable by its combination of paralyses with atrophy, in particular of the tongue. The similar combination of palsies in pseudobulbar paralysis is not associated with atrophy, but there may be slight hemiplegia or diplegia; sometimes a history of repeated attacks. Paralysis of one-half of the tongue and of the opposite arm and leg—a form of crossed paralysis—indicates a lesion of the medulla at the level of the hypoglossal nucleus on the side on which the tongue is paralyzed and wasted. Paralysis of half the tongue, usually with atrophy and associated by implication of the spinal accessory with paralysis of the palate and vocal cord on the same side (Hughlings Jackson), points to lesion of the nerve within the skull or in the upper part of the neck. The prognosis depends on the seat and character of the lesion. As a rule, it is unfavorable; the likelihood of improvement is slight, even in the syphilitic cases.

Spasm of the tongue is an incident of the epileptic convulsion and of chorea. It occurs also as a rare phenomenon in hysteria.

DISEASES OF THE SPINAL CORD.

I. SPINAL MENINGITIS.

Two varieties are usually mentioned—leptomeningitis, inflammation of the pia, and pachymeningitis, inflammation of the dura. Syphilis causes a meningomyelitis in which both membranes may be involved. Tuberculous meningitis of the cord is very rare, unless associated with the same affection of the brain. A meningomyelitis is caused by spinal caries, and in these cases the cord as well as the membranes is involved. Cerebrospinal fever is an acute infectious disease which involves the membranes of both the brain and cord. There is an affection called hypertrophic pachymeningitis, especially of the cervical region, in which the dura is much thickened and the cord

more or less involved; it is sometimes caused by trauma, but in many cases the causation is not clear. Purulent meningitis of septic origin is common; sometimes such an infection extends from the membranes of the brain, as after an otitis media; or from the pelvis, as in the puerperium; or from bed-sores. A pneumococcus infection occurs.

Pathology.—The membranes are congested, thickened, and sometimes covered with a fibrinous or purulent exudate. The nerve-roots are often implicated, and the cord itself may be involved in various degrees. The offending microbe can often be isolated after a lumbar puncture.

Symptoms.— Irritation of the nerve-roots is an early symptom; hence there are pain, stiffness of the back, opisthotonos, contractures of muscles, and even slight clonic spasms. Kernig's sign is usually present. Later in the case there may be pressure symptoms, as paralysis and anæsthesia. In very acute cases there may be chill and fever.

Pachymeningitis cervicalis hypertrophica is an affection almost *sui generis,* especially as caused by trauma. It may closely resemble syringomyelia. There is flaccid atrophic paralysis of the shoulders and arms, spastic paralysis of the lower limbs, and, in some cases, the dissociation symptom, that is, abolition of the pain and thermal senses with preservation of the tactile sense. There may also be pain and stiffness about the neck.

Diagnosis.—It is scarcely possible to distinguish a pure meningitis from a meningomyelitis; the symptoms of disease of the membranes are likely to be associated with some evidence of implication of the cord. Where symptoms of irritation predominate, as pain, stiffness, hyperæsthesia, etc., we may suspect that the membranes are the more concerned. Later, when paralysis and anæsthesia with incontinence appear, we interpret these symptoms to mean that the cord is involved. The eccentric pains, felt at points in the chest or abdomen, may suggest some deep-seated visceral disease, but the diagnosis can usually be made from the associated symptoms. The determination of syphilis as a cause of meningitis is always of first importance. The history of the case may point that way, but it is not always to be relied on, especially when it is negative. The Wassermann and other laboratory tests of the cerebrospinal fluid are more nearly conclusive.

II. MYELITIS.

This term should be restricted to true inflammation of the spinal cord. In the past, however, it has been loosely used for a variety of lesions, such as softening and the destructive effects of trauma. When the term is properly restricted it will be found that genuine myelitis is not a common affection.

Etiology.— This disease is doubtless due in every case to some form of infection or toxæmia. The old ideas that it was caused by exposure to cold, to worry, to sexual excesses, and other such far-fetched notions are no longer credible. At most, cold can act but as a predisposing cause. It is even doubtful whether alcohol causes myelitis. Syphilis frequently causes a meningomyelitis, but this is such a distinct affection that it is treated under a separate heading. Among the causes assigned are the infectious diseases, such as septic infection, varicella, gonorrhœa, and measles. It is claimed that malaria may cause it. But except in the case

of sepsis all these causes are very doubtful. Typhoid fever and smallpox may cause multiple neuritis, which might be mistaken for myelitis by a careless observer. In fact, these two latter diseases must be carefully distinguished. A destructive myelitis may be caused by spinal caries of tuberculous origin, but otherwise tubercle does not often attack the cord. Injury may cause extensive lesions in the spinal cord, and these may become secondarily infected, but they are not primarily inflammatory, nor do they become so in every case. Cancer of the vertebra may also cause myelitis. There are also special forms of myelitis, such as the anterior poliomyelitis, or inflammation of the anterior horns of the gray matter, but these affections are described apart. Finally, cases occur in which acute transverse softening is found post mortem, suggesting the idea of acute infection, but the cause is obscure. In fact, not a few cases of myelitis, and myelitic softening, cannot be satisfactorily accounted for, unless they can be traced to syphilis.

The spinal membranes are often involved in cases of myelitis, so that in effect the condition is one of meningomyelitis, and the cause, whatever it be, may act primarily on the membranes.

Pathology.—This disease may be transverse, focal, disseminated, or diffused. In transverse myelitis the lesion implicates the whole thickness of the cord, but it may be comparatively limited in its upward and downward extent; in fact, not more than one, two, or three segments may be involved. Focal myelitis, described by some authors, is much more rare; as the name implies, the lesion is discrete; and in the disseminated variety there are more than one such lesion scattered in various places in the cord. Diffused myelitis is merely that variety in which the inflammatory process is more widely and continuously extended. The inflamed area may be variously discolored; in some cases it is congested and bright red or pink, in others rather yellowish, in others white. Its consistence varies, but it is usually softer than the normal cord, and it may be so soft as to flow out under the knife. This is the condition often called "white" or "red" or "yellow" softening. The color is merely due to the elements of the blood contained in the softened area. The tissue is usually necrotic, but it is not necessarily purulent. Pus-cells may, however, be found. The membranes may or may not be involved, congested, and thickened, and there are cellular infiltration and thickening of the blood-vessels. Under the microscope the lesions are often found to be characteristically syphilitic, with infiltration of lymphocytes, thickened walls of blood-vessels and pia mater, and secondary sclerosis of medullary tracts.

Symptoms.—It is best to describe the symptoms of myelitis according to the level at which the lesion is located.

If the lesion is in the *cervical region,* all the functions of the cord below that point may be partly or entirely involved. There will be spastic paralysis in the lower limbs, exaggerated knee-jerks and other reflexes, ankle clonus, and Babinski's reflex; incontinence of urine and fæces, if the lesion is transverse or even extensive; anæsthesia to all modes of sensation; possibly bed-sores; in the upper limbs also spastic paralysis, unless the anterior horns of gray matter in the cervical enlargement are involved, in which case there may be, especially in chronic cases, extensive muscular atrophy, even

with fibrillation, in the shoulders, arms, and hands, with flaccid paralysis. Anæsthesia is also more or less complete in the trunk, arms, and hands, and the muscles of respiration may be involved. If the lesion extends to or above the fourth segment there is danger of death from involvement of the phrenic nerve. There may be a girdle sense in the neck or upper part of the chest. As a rule, pain is not an urgent symptom, unless the membranes and nerve-roots are implicated. The pupils may be dilated or contracted, according as the oculopupillary centre is irritated or paralyzed. A total transverse lesion abolishes all functions below its level, but such a lesion in the neck is seldom compatible with prolonged life.

In the *dorsal region* the lesion causes symptoms in the trunk and lower limbs only. There is likely to be a spastic paraplegia, sometimes with contractures, exaggerated reflexes and clonus, Babinski's sign, incontinence of urine and fæces, anæsthesia to all modes of sensation, and a girdle sense about the trunk, or pain radiating through the chest or abdomen, according to the exact level of the inflammation. There may be a zone of hyperæsthesia marking the upper limits of the lesion and caused by irritation of the membranes or nerve-roots. Bed-sores of an aggravated type may form. If the lesion is totally transverse these symptoms are absolute; if, however, the lesion involves only certain structures of the cord, the symptoms may vary within wide limits. Thus, motion may be more involved than sensation; control of the bladder may not be entirely lost; the patient may be able to walk with a weak and spastic gait; and pain may not be urgent. In spinal caries the paralysis is often more motor than sensory, due to the bone lesion being located in front of the cord.

In the *lumbar region* myelitis causes an atrophic or flaccid paralysis of the lower limbs, due to the fact that the anterior horns of gray matter are involved. There is also paralysis of the bladder and rectum in grave cases, bed-sores, pain in the legs, loss of sensation in the lower trunk and lower limbs, and abolished knee-jerks. But here as elsewhere the symptoms will vary somewhat with the extent of the lesion.

The course of myelitis may be acute or exceedingly chronic. The patient may recover up to a certain point, and then remain more or less permanently crippled. Entire recovery is rare. A fatal result is not uncommon. In the myelitis of Pott's disease a good recovery is sometimes obtained.

Diagnosis.—Myelitis is to be distinguished especially from locomotor ataxia, syringomyelia, and multiple neuritis. It may also closely resemble some forms of syphilis of the cord. In fact, syphilis is the commonest cause of myelitis, or meningomyelitis, and the laboratory tests for this disease should always be made. When myelitis is due to trauma or spinal caries the cause is usually apparent.

From locomotor ataxia it is distinguished by the mode of onset, which is usually much more abrupt. There is absence of the fulgurant pains, the peculiar ataxic gait, the swaying with closed eyes, the abolished knee-jerks (unless in lumbar myelitis, in which case, however, there is flaccid paralysis and muscular atrophy in the lower limbs, very different from tabes). There is also absence of the Argyll-Robertson pupil, with optic atrophy; and the paralysis of the bladder and bowel is much more complete and of earlier onset than in locomotor ataxia.

In syringomyelia the symptom-complex is quite different from ordinary myelitis. There is especially the dissociation symptom, in which the temperature and pain senses are abolished without impairment of tactile sensation; also scoliosis, arthropathies, and vasomotor changes. The onset is also more gradual than in myelitis; the bladder and bowel are not so likely to be involved. Still, some cases of traumatic meningomyelitis of the cervical cord closely resemble syringomyelia, and are distinguishable only with care. The history of trauma in these cases is significant.

In multiple neuritis the symptoms are distinctly peripheral. The nerve-trunks are involved, and are often painful on pressure. The muscular masses, especially the calf, are exquisitely sensitive. The paralysis is flaccid, and the muscles waste. The reactions of degeneration may be present. The deep reflexes are abolished. The anæsthesia, while present in varying degrees, is not always seen in such extensive areas as in myelitis. The bladder and bowel, as a rule, are not involved. There may be mental symptoms—the so-called Korsakoff's psychosis—and there is a history of exposure to alcohol, to lead, to arsenic, or to diphtheria. It is sometimes difficult at the bedside to say whether or not a myelitis is due to syphilis, but in some forms of syphilis of the cord the type is quite distinct. In some cases of cord-syphilis, there may be, or may have been, some involvement of the cerebrum or cranial nerves. Lumbar puncture and the appropriate tests of the cerebrospinal fluid will often throw light on the causation and pathology of these cases.

The constitutional reaction in myelitis at the beginning is not marked, except in the acute anterior poliomyelitis of children. There may, however, be some fever and weakness of the pulse. In advanced cases, with bed-sores and incontinence of urine, symptoms of sepsis may occur. Infection of the bladder is always a grave complication. It may be due to the use of the catheter.

III. ANTERIOR POLIOMYELITIS.

Inflammation of the anterior horns of gray matter in the spinal cord may occur as an acute or a subacute disease. The acute form is usually a disorder of childhood, although adults are not entirely exempt. In children it is called *infantile paralysis*. The subacute form is rare, and is usually seen in adults. Progressive muscular atrophy, or chronic anterior poliomyelitis, is also a disorder of adults, but its inflammatory nature may be doubted. It is a slowly progressive degeneration of the ganglion cells in the anterior horns.

The cause of acute anterior poliomyelitis has now been clearly demonstrated to be an infection or toxæmia. Infantile paralysis has been known to prevail as an epidemic, as in the instance reported by Medin, in which 44 cases occurred within a few weeks in one town in Sweden. Epidemics have been observed in the United States. A series of 126 cases occurred in Rutland, Vt., in 1894. It has prevailed epidemically in many parts of the world in the last 30 years. Among the great epidemics have been those in Norway, in Vermont, in Queensland, in Vienna, in Wisconsin, in

Westphalia, and in Nebraska.[1] An epidemic, or very prevalent type, of the disease occurred in New York City in the summer and autumn of 1907, and the affection has since prevailed epidemically in other parts of the United States. In Philadelphia and vicinity an extensive epidemic occurred in 1916. Isolated cases sometimes follow the infectious diseases, as measles, scarlet fever, whooping-cough, and diphtheria.

We owe to the researches of Flexner and Lewis[2] and the later experiments of Flexner and Noguchi[3] the demonstration of the infectious nature of epidemic anterior poliomyelitis and its exciting cause. The microorganism consists of filterable globoid bodies measuring from 0.15 to 0.3μ in diameter and arranged in pairs, chains or masses. Inoculation into the brain or the sciatic nerve and peritoneal cavity of monkeys is followed after an incubation period of forty-eight hours to several weeks by the clinical symptoms and pathological effects characteristic of epidemic poliomyelitis.

Pathology.—Flexner says that the vascular lesions are the primary causes of the lesions of the central nervous system. In the early stage there is hyperæmia of the horns and some congestion of the cord; the ganglion cells are swollen and indistinct; the vessels are engorged, and white cells are seen migrated from them. Later the ganglion cells are atrophied or even entirely destroyed; the nerve fibrils are obliterated, and the horns are reduced in size. The disease may be located in either the cervical or lumbar enlargement. In the subacute form the lesions may be found more extensively spread; the anterior horns in many parts of the cord are involved, and in some cases the neighboring white matter is invaded.

Symptoms.—Infantile paralysis begins abruptly, with constitutional reaction. There is fever, headache, sometimes moderate stupor and delirium, and convulsions may occur. Some days may elapse before the true nature of the disease is recognized, and then it is found that the child is paralyzed in one or more limbs. This paralysis is flaccid, with abolished reflexes, but in the very early stage, with acute meningeal symptoms, the reflexes may, according to Weisenberg, be active, and Kernig's sign may be present. Pain is not prominent and may even be entirely absent; but in some cases it may be severe in the early stages. The initial palsy may involve the whole limb, but as time passes a partial recovery takes place, until finally the paralysis is located, and remains stationary, in a few muscles or a group of muscles. There is no anæsthesia in the paralyzed part, as a rule, to which there may be exceptions. The bladder and bowel are not involved, except in grave cases. Infantile palsy is a common cause of club-foot, the type of deformity depending upon the group of muscles paralyzed. The reactions of degeneration are present, and the muscles do not show fibrillation. In the arm the muscles oftenest damaged are the deltoid, biceps, brachialis anticus, and supinator longus. Deformity of the hand is not common. The paralyzed limb does not grow normally, but remains partially stunted and even shortened. In the recent epidemic in New York bulbar symptoms were sometimes seen, even in mild cases in which recovery ensued. Meningeal symptoms, such as pain, rhachialgia, and rigidity, were marked, and some-

[1] Collins (Trans. Ass. Am. Physicians, Vol. XXV, p. 90) gives statistics of the various epidemics in tabulated form.

[2] Flexner and Lewis's articles are in vols. 53, 54 and 55 of the Journal of the Am. Med. Ass.

[3] Flexner and Noguchi. Trans. Ass. Amer. Physicians, Vol. XXVII, 1913.

times there was photophobia. Nothing was found in the cerebrospinal fluid or in the blood to show the cause, although the disease had some resemblance to epidemic cerebrospinal meningitis. According to Harbitz and Scheel, the brain and bulb were often involved in epidemics in Norway. Many fatal cases occur in the epidemic form.

Wickman has attempted to establish various types of the disease, such as the spinal poliomyelitic type, the type of ascending or descending palsy (Landry's paralysis), the bulbar and pontine type, the cerebral (encephalitic) type, the ataxic type, the polyneuritic type, the meningeal type, and abortive types. But the spinal type is the predominating one; all other forms are rare varieties.[1] A transverse myelitic type, with paralysis and anæsthesia below a certain level, and with loss of sphincter control, is a very rare form.

The subacute form of this disease is also a grave affection. Duchenne recognized an *ascending* and a *descending* type. In the former the disease begins in the lower extremities, and later invades the upper limbs; in the latter the course is the reverse; but some of Duchenne's cases may have been instances of Landry's disease. The paralysis is characteristic of a lesion of the anterior horn: the muscles become atrophied, and they may show fibrillation. Bramwell points out that the paralysis precedes the atrophy, that the reflexes are abolished, and the reactions of degeneration are present. In cases in which recovery occurs there may be permanent paralysis in some muscles. Sensory and bladder symptoms are wanting. Bulbar symptoms have been reported. The disease is rare, and it may not be due to the same infecting agent as the acute form. Flexner suggests that there may be more than one form of the infection.

Diagnosis.—Infantile paralysis in its initial stage may be mistaken for an acute febrile infection, or infantile convulsions, and the paralysis may be overlooked for several days. After the paralysis is noted the disease is not likely to be mistaken. The flaccid palsy, the abolished reflexes, the onset of wasting, all coming on after the symptoms of an acute infection, without anæsthesia, and limited to one limb or part of one limb, are unmistakable. The disease may also resemble multiple neuritis, which is sometimes seen in children; but multiple neuritis is more wide-spread and symmetrical, pain in the affected limbs is more common, the cause may be traced in some poison, and complete recovery may occur. Nevertheless, there is a polyneuritic type of poliomyelitis which may not be easy to distinguish clinically. The subacute form in adults bears a resemblance to multiple neuritis, but the fibrillation in the muscles, and the absence of sensory symptoms and of pain on pressure over nerve-trunks and muscles, serve to distinguish it. The reactions of degeneration may not be so promptly established as in multiple neuritis. The resemblance of poliomyelitis to Landry's paralysis may be close; but the latter is a more acute affection, and the tendency to a fatal ending is more marked. Fibrillation is not seen in Landry's disease, but neither is it reported in all cases of inflammation of the anterior gray horns. The two affections have not a little in common and some observers believe that they are identical. In myasthenia gravis there is the rapid exhaustion on exertion, and the affected muscles do not atrophy

[1] Report of the Collective Investigation Committee, N. Y. Epidemics, pp. 50, 51.

or show fibrillation. In all these three affections, namely, poliomyelitis, Landry's paralysis, and myasthenia gravis, the exact causation and pathology have points of resemblance; they seem to depend on a poisoning of the motor neurons, or, in the case of myasthenia gravis, on some affection of the muscular fibres, and the motor symptoms are not altogether dissimilar. Poliomyelitis may resemble cerebrospinal meningitis, and has been confounded with it, but in the latter disease the infecting organism can usually be found in the cerebrospinal fluid. A striking difference is seen in the seasonal prevalence, which for epidemic poliomyelitis is midsummer, and for the other disease is late winter and early spring. Flexner points out changes in the cerebrospinal fluid in poliomyelitis which may have diagnostic significance. The fluid first contains a number of small cells hardly larger than lymphocytes, but showing a polyform nucleus, a few lymphocytes and red cells. Later the white cells increase; at the end of 72 hours a large number of mononuclear cells appear, and the fluid is opalescent. On the day of the paralysis the fluid tends to be only cloudy and contains a mixture of large and small mononuclear cells. Later investigators attach great importance to these and similar changes in the cerebrospinal fluid.

IV. ACUTE ASCENDING PARALYSIS.

This disease was first described by Landry in 1859, and is usually called by his name. It was for a long time depicted as a disease without a pathology, but the more refined methods of recent days have tended to make it out an affection of the peripheral motor neurons.

Pathology.—The pathology of Landry's paralysis is not established. Some observers have found changes in the ganglion cells in the anterior horns of gray matter and in the bulb, as chromatolysis and swelling of the axis-cylinder, also foci of inflammation and capillary hemorrhages in the cord and medulla oblongata. Changes have been found in the peripheral nerves. As said above, the tendency now is to identify this disease with acute poliomyelitis.

Symptoms.—The disease usually begins as a flaccid paralysis in the lower limbs, and extends upward, involving the muscles of the trunk, the arms, and finally the bulb. Anæsthesia is not present, although there may be some slight dulness or retardation of sensation. The muscles do not waste perceptibly, but in rapidly fatal cases there is not time. The electrical reactions may be preserved. There is no incontinence, as a rule, although exceptions occur. In some cases the initial symptoms are in the bulb and upper extremities—the descending type. The mind is not affected. Fever is not common. There may be hyperidrosis. The course of the disease is sometimes very rapid. Death results from exhaustion and asphyxia. A few recoveries have been claimed.

Diagnosis.—Landry's paralysis resembles a rapidly fatal multiple neuritis, except that the sensory symptoms are almost altogether wanting, but if the pathology be proved to be a toxic affection of the motor neurons, it is rather like acute poliomyelitis, with which some observers identify it. It is not likely to be confounded with any other disease, unless it be with myasthenia gravis, in which the history and course are different and the

exhaustion symptom is marked. It is possible, however, that in myasthenia gravis there may be some involvement of the motor neurons. A careful study of the cerebrospinal fluid in Landry's paralysis may serve possibly to establish its identity with poliomyelitis.

V. PROGRESSIVE MUSCULAR ATROPHY.

There is a degenerative process in the spinal cord, usually chronic, which affects chiefly the anterior horns of gray matter, especially in the cervical region. This is the disease called, from its anatomy, chronic anterior poliomyelitis; and, from its clinical form, progressive muscular atrophy.

Pathology.—Chronic anterior poliomyelitis is a destructive process which invades the anterior horns of gray matter. It has the appearance under the microscope of being a degeneration of the motor neurons. Thus the large ganglionic cells are shrunken or even destroyed, the interlacing nerve fibrils of the anterior horns are obliterated, and these horns are smaller than normal. But there are cases in which this identical process is found, and in which is seen in addition a degeneration of the lateral, or crossed, pyramidal tracts. Where this association exists the clinical form differs, for in addition to muscular atrophy there is seen a spastic paralysis with exaggerated reflexes. This latter affection has been given a distinct name—amyotrophic lateral sclerosis—and is described in most text-books as a distinct disease. Some neurologists believe these two so-called diseases are merely different forms of the same pathological process. For the sake of convenience and to conform to custom the two affections will be described here separately. These two clinical forms represent disease of the motor neurons: in anterior poliomyelitis it is the neurons of the lower order which are affected, whereas in amyotrophic lateral sclerosis not only the neurons of the lower but also those of the upper order are involved.[1] The possible rôle of syphilis as a cause of these affections has been suggested. Nonne describes a chronic anterior poliomyelitis due to syphilis.

Symptoms.—The disease usually begins with wasting of the muscles of the hands, especially of the thenar and hypothenar groups, and the interossei. Later the muscles of the forearms become involved, then those of the upper arms and shoulders. This is called the Aran-Duchenne type. Other forms appear, as, for instance, early wasting of the deltoids, supra- and infraspinati, and biceps—the upper arm type. In some rather rare or advanced cases the muscles of the lower limbs are involved. The neck and trunk muscles may also waste. Various deformities occur, such as the claw hand, or "main en griffe," in which the proximal phalanges are overextended and the distal phalanges are flexed; and the so-called monkey hand, or "Affenhand" of the Germans, the "main de singe" of the French, which is caused by overaction of the long extensor of the thumb, causing the metacarpal bone to be displaced backward and to lie in the same plane as the metacarpal bones of the fingers. In the lower limbs various forms of club-foot result. As the wasting progresses, loss of power occurs, until in advanced cases the wasted arms hang powerless at the sides. In the

[1] R. T. Williamson: "Amyotrophic Lateral Sclerosis and Progressive Muscular Atrophy," Edinburgh Med. Journal, April, 1917, p. 304.

FIG. 390*a*.—Progressive muscular atrophy.—Atlas of Clinical Medicine (Dr. Byrom Bramwell). By courtesy of the author.

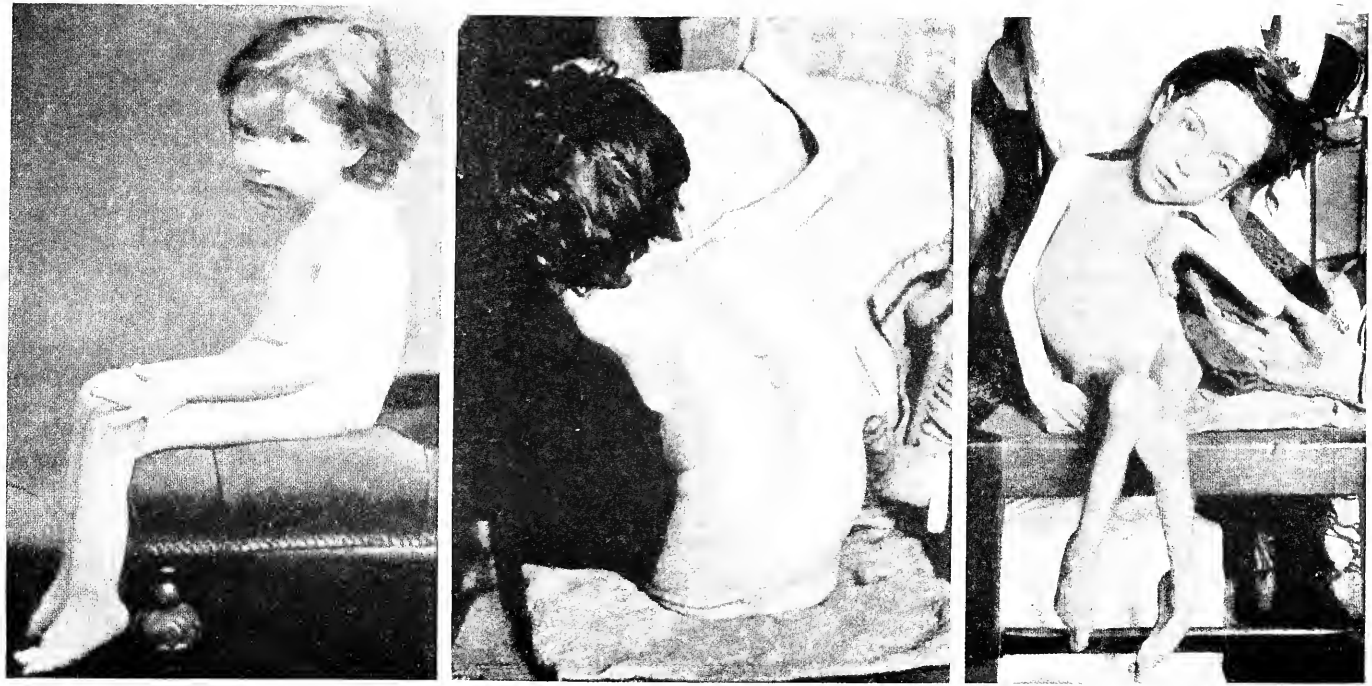


FIG. 390*b*.—Muscular atrophy in a child.—Atlas of Clinical Medicine (Dr. Byrom Bramwell). By courtesy of the author.

pure forms, that is, in cases in which the lateral tracts are not involved, there is no spastic paralysis, and the reflexes, either of the arms or legs, are not increased. In advanced cases the reflexes in the affected muscles may be much diminished or even lost; but this is not invariable, for even in very much wasted muscles a sharp tap will sometimes elicit a slight response. The affected muscles are the seat of fibrillary twitchings. The complete reactions of degeneration are not present, although some modal change and partial reaction may be seen in advanced cases. There is no anæsthesia of any kind, nor are the sphincters usually affected. Scoliosis does not occur. In cases in which the neck muscles are greatly wasted there may be head-drop, but this symptom occurs more frequently in amyotrophic lateral sclerosis. As a rule, there are no bulbar or oculomotor symptoms. The disease may be very chronic, lasting for many years. Some aberrant forms are seen, but the above are the most common types. The disease, as a rule, begins in adult life. It is progressive and incurable.

Diagnosis.—The distinction has already been made clear between this affection and amyotrophic lateral sclerosis. In the latter disease there is spastic paralysis in addition to the muscular atrophy, and this spasticity with exaggerated reflexes is seen in the wasted arm muscles as well as in the lower limbs, in which there may be little or no wasting. In pure cases of progressive muscular atrophy there is no real spasticity in the affected muscles and no spastic paralysis of the lower limbs. It must be acknowledged, however, that the dividing line between the two diseases is not always sharply defined. In progressive muscular atrophy increased reflexes with some spasticity of the limbs are sometimes observed. The disease may resemble syringomyelia, but is to be distinguished by the absence of the dissociation sensory syndrome, and of the trophic lesions, arthropathies, and scoliosis. From pachymeningitis of the cervical cord it is to be distinguished also by the absence of sensory symptoms and of pressure palsies of the cord. From multiple neuritis it is distinguishable by its history and course, the absence of sensory symptoms, the distribution of the muscular atrophy, the absence of reactions of degeneration, the fibrillary twitchings, and the preserved or even increased reflexes. The various muscular dystrophies often resemble progressive muscular atrophy of spinal origin. They usually begin in childhood, however, and the muscles do not present fibrillary tremors, nor are the reflexes preserved. They are sometimes hereditary and more than one case may occur in a family. In some cases of myelitis there are muscular atrophies and spastic paralysis, but the onset is more rapid and the course of the disease more acute; sensory symptoms are present, and there is paralysis of the bladder and rectum. In the advanced stages of locomotor ataxia there is sometimes seen extensive muscular atrophy, but the history of the case, the fulgurant pains, the ataxia, the abolished knee-jerks, the atony of the bladder, the optic atrophy, the Argyll-Robertson pupil all serve to distinguish tabes. So, also, in Friedreich's ataxia there may be some degree of muscular atrophy in later stages of the disease, but the history and the typical symptoms, as ataxia, nystagmus, and the affection of speech, will prevent error. As already said, syphilis of the spinal cord may present progressive muscular atrophy.

VI. AMYOTROPHIC LATERAL SCLEROSIS.

Amyotrophic lateral sclerosis may be said to be a progressive muscular atrophy *plus* a lateral sclerosis. The two affections may be syndromes of one and the same pathological process, depending for their differences upon a mere difference in the distribution of the lesions in the cord. Amyotrophic lateral sclerosis was first described by Charcot, and is sometimes called by his name.

Pathology.—The lateral tracts are sclerosed, sometimes throughout their entire length in the cord, the process stopping in the medulla; but Alfred W. Campbell has traced the degeneration as far as the brain cortex, where it is seen especially in the large Betz cells in the motor region—that is, the ascending frontal convolution and paracentral lobule. The degeneration of the anterior horns in the cervical region is marked, and sometimes the gray matter in the medulla oblongata is involved.

Symptoms.—The disease begins as a muscular atrophy, most marked at first in the hands, but gradually including the arms, shoulders, neck, and even the trunk. In this respect its appearance is like progressive muscular atrophy. The essential muscles of the hands are much involved, and "claw hand" or "monkey hand" may develop; the arms become so powerless that they hang useless at the sides; the muscles of the neck atrophy and permit the head to fall forward on the chest, and when the patient raises the head it goes up into place with a quick jerk somewhat like the closing of a blade of a penknife; in rare cases bulbar symptoms occur, the patient having difficulty in swallowing and the voice having a nasal twang. Fibrillary twitchings are present. The muscular atrophy seldom invades the lower limbs. Along with the atrophy appear the evidences of sclerosis of the lateral tracts. Even in the wasting muscles there is hypertonus, and the reflexes are increased. The paretic arms may be slightly spastic. The biceps-jerks, triceps-jerks, and wrist-jerks are exaggerated. The spastic paresis of the lower limbs is very marked: the patient walks with a feeble but spastic gait, the feet scraping the floor. The reflexes are all exaggerated, such as the knee-jerks and ankle-clonus, and there is seen the plantar extensor reflex of the great toe—the Babinski reflex. There is no anæsthesia, nor any paralysis of the bladder or bowel as a rule.

FIG. 391.—Amyotrophic lateral sclerosis, showing head-drop.—Lloyd.

Diagnosis.—The rules for differential diagnosis are practically the same as in the case of progressive muscular atrophy. These two diseases shade into each other and may be only variants of the same morbid process. Dejerine

claims that bulbar symptoms do not occur in true progressive muscular atrophy, and that they are indicative of amyotrophic lateral sclerosis. The latter disease runs a more rapid course than the former. The increase of the deep reflexes, the spastic gait, and the paralysis of the muscles before the onset of atrophy are in favor of amyotrophic lateral sclerosis. The possibility that syphilis may be a cause has been suggested, and this problem needs further investigation.

VII. PRIMARY LATERAL SCLEROSIS.

Some authors, as Charcot, Erb, and Dreschfeld, have described a sclerosis of the lateral tracts which appears to be primary, that is, it does not depend upon a focal lesion, and is not associated with degeneration of other structures or tracts of the cord.

Symptoms.—The symptoms are the same as those already described for amyotrophic lateral sclerosis, *minus* the muscular atrophy. There is spastic paralysis of the legs, with "clasp-knife rigidity," in which the limb, when passively extended, resists, then suddenly yields, as in the closing of the blade of a knife. The arms are sometimes involved. The deep reflexes are all exaggerated. There is no anæsthesia, nor paralysis of the bladder or bowel. Lloyd and Ludlum have described a series of cases occurring in syphilitic subjects under the head of *essential* lateral sclerosis.

Diagnosis.—It is particularly necessary to eliminate every focal lesion from which lateral sclerosis could occur as a descending degeneration, and every disease of other structures than the lateral tracts. The affection is rare, and thus far partakes rather of the nature of a pathological curiosity. Mills has described an ascending hemiplegia which goes by his name, and which has its origin in the cord; the lateral tract of one side is especially involved. It is probably distinct from the affection here described. Spinal syphilis may simulate this disease, especially when the specific lesion is confined to the lateral aspects of the cord. It may also be simulated in the early stages of multiple sclerosis. It is to be distinguished from some degenerative brain and cord affections; as that described by Strümpell, in which spastic paralysis occurred in several members of the same family; and from spastic diplegia in children, the result of focal lesions in the brain, in which cases there is likely to be mental defect.

VIII. LOCOMOTOR ATAXIA.

This disease, also called tabes dorsalis, is characterized by a degeneration of the posterior columns of the spinal cord. It has been recognized only within comparatively recent years, and was first described clinically about 1840 by Romberg, who, however, did not recognize its morbid anatomy. Todd, of England, and Cruveilhier, of France, were among the first to associate it with disease of the posterior columns.

Pathology.—There has been much discussion as to the exact seat of the initial lesion in tabes. Vulpian, Charcot, and others held that it was primarily an inflammation of the posterior nerve-roots. Others, as Marie, Marinesco, and Wollenburg, contend that the ganglion cells in the posterior ganglia are the first involved. Leyden and Goldschneider hold that the

starting-point is in the peripheral sensory nerve endings; while still others, as Nageotte, Redlich, and Obersteiner, believe that the disease begins as a meningitis, affecting especially the posterior nerve-roots. Ferrier concludes that none of these theories is satisfactory, but that the essential lesion of tabes is a dystrophy, similar to that induced by certain toxic agents, affecting the sensory protoneuron as a whole. The disease, however, is not confined to the spinal protoneuron, but may affect the optic, the sympathetic, and certain motor neurons.

When fully established locomotor ataxia shows a degeneration especially marked in Goll's columns in the cervical region, in Burdach's columns more or less marked at various levels of the cord, and in the lumbar region in the areas known as the *bandelettes externes* of Pierret. There is a rare cervical type in which the columns of Goll in the neck entirely escape. There is also optic degeneration, and in advanced cases muscular atrophy. The posterior nerve-roots are degenerated, and there is leptomeningitis. Syphilis is now recognized as the invariable cause of tabes. Noguchi found the spirochæte in the disease, and the Wassermann and other laboratory tests are confirmatory.

Symptoms.—Locomotor ataxia is known by an ataxic gait, loss of static equilibrium, abolished knee-jerks, crises and fulgurant pains, sensory changes, atony of the bladder, loss of sexual power, optic atrophy, and the Argyll-Robertson pupil. Other but rare symptoms are muscular atrophy, arthropathies, and trophic lesions.

The ataxic gait of tabes is its most conspicuous symptom. It is not due to paralysis but to incoördination. There may be full motor power, even an excessive use of power, but the muscles do not act in harmony with the will. The patient walks with the feet well apart; the foot is lifted high from the ground, thrown out widely, and brought down with a stamp, the heel striking the floor first. It is evident that the patient feels the unreliability of his gait, for he watches the floor, and aids his progress by the use of his eyes. Hence he walks with especial difficulty in the dark and in coming downstairs. He may not be able to walk at all with his eyes closed. The ataxia may also be marked in the arms and hands, as in touching the tip of the nose with the forefinger (with closed eyes), unbuttoning his coat, etc. It is also seen when the patient is lying down and attempts to move his legs. It is often an early symptom, but may be preceded by fulgurant pains and changes in the pupils.

The loss of static equilibrium is seen when the patient attempts to stand without support and with this feet close together; but it is much increased when he closes his eyes. He then sways violently and in some cases would even fall. This is the Romberg symptom.

The knee-jerks are abolished early in tabes. This is one of the most constant symptoms, and is the Westphal sign. The other deep reflexes are also lost. The Achilles jerks are lost early in the disease; even before the knee-jerks, in most if not in all cases.

The fulgurant pains are usually an early symptom. They are lightning-like and severe, and felt most in the lower limbs. They may prevent sleep, and are most urgent in their demand for relief. They are usually paroxysmal, and may remit for days and even weeks. Closely associated

FIG. 332*a*.—Lymphadenoma.—Atlas of Clinical Medicine (Dr. Byrom Bramwell). By courtesy of the author.

with these pains are the girdle sense and various crises. The former is felt as a band tied about the waist or abdomen, or about the chest, or even about one limb. The crises are bouts of pain felt in various parts of the body, especially in the epigastric region, but sometimes in the thorax, the larynx, or even the rectum. They may simulate some disorder of one or other internal organ. The laryngeal crises cause a sense of strangling and excite cough. According to Semon the essential cause is a paralysis of the abductors of the vocal cords, and the attacks may begin with a sense of tickling, or even pain in the throat, followed quickly by a sense of suffocation; among accessory symptoms are dizziness, mental confusion, and even loss of consciousness with convulsive movements, but the attacks are in no sense epileptic. Involuntary passage of urine and fæces is occasionally present.

There are various disorders of sensation. Tactile anæsthesia is not always present, or it may be present in only limited areas; in other cases, especially in the advanced stages, it may be extensive. There may be paræsthesia, or altered sense. Thus, the patient may have abnormal feelings in the soles of the feet, causing him to feel as though he were walking on some soft substance, as velvet or mud. There may also be numbness, or formication, or a sense of cold. Alteration of the thermal sense, however, is not usually marked. Analgesia is common: the patient has lost the sense of pain, particularly in the legs; pinching or sticking with a pin is not felt as pain. There is often loss of muscular sense and sense of position; also of the sense of pressure and sense of active and passive motion; and some writers attach great importance to these changes in deep sensibility as the fundamental cause of the ataxia.

Some loss of power in the bladder is often an early symptom; at first there is difficulty in extrusion, later there may be retention or even incontinence. Loss of sexual power is not uncommon; occasionally, in the early stages, there is sexual excitement.

Optic atrophy is frequent in tabes, but its exact frequency is a subject of some debate. Gowers, in 70 cases of posterior sclerosis, found only 9 with this condition; Voight in 52 cases found 9; and Erb in 56 cases found 7. Optic atrophy may appear early, sometimes before the onset of ataxia. It is primary; that is, it is not dependent on a preceding neuritis. It may progress to complete blindness, but its progress is often slow. Changes in the pupils are likely to be early symptoms of tabes. The sympathetic reflex from irritating the skin of the neck is often lost. There may be myosis, sometimes extreme; the pupils are contracted. Sometimes they are unequal, and even irregular in outline. Later in the disease they may be widely dilated. But the commonest change is the Argyll-Robertson pupil, in which the reflex to light is abolished but the movement on accommodation is preserved.

The exterior muscles also of the eyes are sometimes involved. Thus, there may be ptosis of one or of both upper lids. Other paralyses of the third nerve and of the sixth are also seen. The third nerve may be paralyzed in only some of its branches, causing a partial oculomotor palsy; and this may tend to disappear and reappear.

Among the rarer symptoms of tabes are arthropathies. These may affect the knee, ankle, hip, elbow, or shoulder. A very typical form is that seen in the knee. The joint is the seat of a painless swelling; there is denudation of the articular surfaces, grating, effusion of fluid, osteophytes, and deformity. The joint is relaxed, allowing the knee to be overextended or bent backward. The whole leg may be enlarged and brawny. In the shoulder and hip an atrophic form of arthropathy is seen; there is preternatural mobility. The head of the humerus or femur may be entirely destroyed, as shown by the X-ray. Sometimes painless fractures occur. A condition known as hypotonia exists: the joints may allow a much wider range of motion than normal. Thus, when the patient lies upon his back, the whole lower limb, straightened at the knee, may be so extended that, in extreme cases, the foot may even rest alongside the neck. A characteristic trophic lesion is the *mal perforans*, or perforating ulcer. This forms on the ball of the foot or great toe; it is deep, painless, and obstinate in healing. Muscular atrophy is seen in some advanced cases of posterior sclerosis, and may be extreme. It is probably dependent on involvement of the anterior nerve-roots or anterior horns, or on a peripheral neuritis.

FIG. 392.—Arthropathy of the left ankle-joint in locomotor ataxia.—Lloyd.

The course of tabes is usually chronic. It is a disease of long duration, often extending over many years. In the advanced stages the patient is unable to leave his chair, or even his bed, the victim of painful crises, partially or entirely blind, with incontinence of urine and possibly with one or more arthropathies. There is a *sensory type* of tabes. This is marked by early optic atrophy, proceeding to complete blindness, associated with severe lancinating pains and crises, with lost knee-jerks, but without impairment of gait. This form may persist for many years, finally developing ataxia. Buzzard reported a case which preserved this type for fifteen years, and Gowers mentions one in which optic atrophy had existed for twenty years before the onset of incoördination. This type is peculiarly liable to arthropathies. Locomotor ataxia sometimes coexists with general paresis. It may precede that disease, as is the more common way, or in some cases it may follow it.

There is a juvenile tabes, which is the result of hereditary syphilis. The disease, however, is usually one of adult life, the initial symptoms generally showing themselves between the ages of 30 and 40 years. It is not limited to any race or country, but is seen wherever syphilis abounds; and the statement that locomotor ataxia does not occur in the negro race, is erroneous. It is not so common among women in any race as among men. It has been seen in both husband and wife.

Diagnosis.—Locomotor ataxia is to be distinguished from multiple neuritis by the fulgurant pains, the crises, the pupillary changes, the bladder symptoms, and the absence of true paralysis with atrophy and the reactions of degeneration. There is a *pseudotabes* due to multiple neuritis, in which ataxia is marked, but the history of the case, the flaccid paralysis, with atrophy and electrical changes, the painful nerve-trunks and muscles, as well as the absence of the optic atrophy and pupillary changes, serve to distinguish it. Muscular atrophy with loss of power may occur in advanced stages of tabes, but the other tabetic symptoms and the history of the case should prevent error. From syringomyelia, in which there sometimes occurs an ataxic type, tabes is distinguished by the absence of the dissociation syndrome, of the scoliosis, of the spastic paralysis in the legs with exaggerated knee-jerks, and of the muscular atrophy in the shoulders and arms. Arthropathies occur in both diseases. Optic atrophy is seldom if ever seen in syringomyelia.

Progressive muscular atrophy and amyotrophic lateral sclerosis are hardly to be confounded with tabes. The muscular atrophy, often with preserved or even exaggerated reflexes, and the absence of ataxia, crises, optic atrophy, and the Argyll-Robertson pupil, are sufficient to distinguish the one from the other. From multiple sclerosis tabes is distinguished by the ataxia, which is not the same as the intention tremor of the former disease, in which the movement is jerky, tremulous, and very marked only on voluntary motion. Moreover, in multiple sclerosis there is usually a spastic gait with exaggerated reflexes, and an absence of fulgurant pains and crises; nystagmus and scanning speech are observed; optic atrophy may be present, but only rarely; the Argyll-Robertson pupil is not seen. The painful crises of tabes, occurring in the chest or abdomen, may simulate disease of some internal organ, as angina pectoris or gastric ulcer, or some affection of the bowel, and laparotomies have been performed by surgeons under this mistaken diagnosis. The resemblance is only superficial, and the coexistence of other tabetic symptoms points to the correct diagnosis. The laryngeal crises sometimes simulate laryngismus stridulus, especially in the crowing inspiration at the end of the attack, and even epilepsy, when consciousness is lost and spasmodic movements occur; but the diagnosis is to be made from the associated tabetic symptoms in the pupils, the gait, and the reflexes. In various forms of myelitis there may be a girdle sense, and in inflammation of the lumbar cord loss of the knee-jerks; but usually the knee-jerks are exaggerated, and the optic and pupillary symptoms are not so marked. Nevertheless, in various forms of cerebrospinal syphilis there are often seen pupillary changes, such as abolition of the reflex to light; and there may also be an optic neuritis. There is spastic paralysis in the legs, sometimes incontinence of urine and fæces early in the case, and the general history is different. Crises and fulgurant pains are wanting. Anæsthesia, clearly delimited at its upper margin, is often present. In the meningomyelitis of syphilis we sometimes see an ataxic paraplegia, in which the incoördination is very similar to that of tabes; but it is associated with a spastic state of the lower limbs and exaggerated knee-jerks.

Tabes may coexist with, or lead up to, general paresis, but the peculiar mental symptoms serve to distinguish the latter disease.

Serological changes which are characteristic of syphilis, are seen in locomotor ataxia, and are described elsewhere in this work (p. 647, Vol. I).

IX. ATAXIC PARAPLEGIA.

This is a syndrome caused by a combined sclerosis of the lateral and posterior columns of the spinal cord.

Pathology.—There is a posterior sclerosis very much as in locomotor ataxia, and in addition a sclerosis of the lateral columns of the cord, but more especially of the crossed pyramidal tracts. Occasionally the direct cerebellar tracts are also involved; but it is rather rare for other parts of the anterolateral columns to be invaded, as, for instance, Gowers's tracts or the direct pyramidal tracts. The disease seems to be a system disease, confining itself to certain definite tracts, and not a diffused myelitis. There is a form of syphilitic meningomyelitis, however, which closely resembles combined sclerosis both clinically and anatomically: in this form, the inflammation is located in the lateral and posterior aspects of the cord; but the membranes are involved first and the posterior and lateral columns are affected secondarily.

Symptoms.—Because of its morbid anatomy it is easy to understand that ataxic paraplegia partakes of the nature both of locomotor ataxia and spastic or primary lateral sclerosis. This idea, however, requires some qualification, for those two diseases cause some contrary symptoms, such as lost knee-jerks by the one and exaggerated knee-jerks by the other, and the two symptoms cannot coexist in the same person. As usually seen, the share of the disease contributed by the posterior sclerosis is the ataxia. There may be some other tabetic symptoms, as lancinating pains, sensory changes, and optic atrophy, but they are rare. Except for the ataxia the disease takes its form largely from the sclerosis of the lateral tracts. There is a spastic gait, with exaggerated reflexes, and these with the ataxia produce a rather confusing picture.

There is a type of the disease in which the tabetic symptoms predominate, and there is then seen the ataxia with lost knee-jerks, fulgurant pains, and bladder weakness, combined with some loss of power due to the lateral sclerosis. According to Oppenheim this predominance of the ataxia and other symptoms of posterior sclerosis is likely to appear in the more advanced stages.

Some authors claim that the muscle tonus is lowered, or at least not increased. This may be so in cases in which the posterior columns are the more involved; but in many cases, and especially in advanced stages, there is increased tonus along with the spastic paraplegia. The type of the case, whether more ataxic or more paraplegic and spastic, will depend upon which region of the cord is more affected.

Diagnosis.—The disease is not likely to be mistaken for any other. The combination of ataxia with a spastic gait is distinctive. Multiple

sclerosis may be simulated by ataxic paraplegia, but in multiple sclerosis there is not a true ataxia, but rather an intention tremor, with nystagmus and speech defects. From some forms of spinal syphilis the distinction is not always easy; in fact, ataxic paraplegia is probably nothing else but a form of spinal syphilis. Diffused myelitis may also resemble combined sclerosis, but the symptoms are not so characteristic of a system disease. The ataxia closely resembles that of tabes dorsalis, but the spastic paraplegia, with exaggerated knee-jerks, marks the difference.

X. HEREDITARY ATAXIA.

This affection, also called Friedreich's disease, is a family rather than an hereditary disorder. It is often seen in several brothers or sisters, but is seldom directly hereditary. It is due to degeneration of several tracts of the spinal cord, especially the posterior columns, but the lateral tracts, and sometimes the direct cerebellar tracts and Clarke's columns, may also be involved. The affection of the posterior columns seems to give the disease most of its individuality.

Symptoms.—The patient has an ataxic or staggering gait, in which he keeps his feet far apart and sways his body violently. The gait is not identical with that of locomotor ataxia; the stamping is not so marked, the swaying of the body is more conspicuous, and the progression has something in it that even suggests a cerebellar lesion. There may also be violent ataxic movements while sitting or even reclining. The swaying is not particularly increased by closing the eyes. Later there are ataxic movements in the arms. The knee-jerks are abolished. Anæsthesia is not present, unless in the advanced stages, and then not always. True paralysis of the legs occurs also in advanced stages, and there may even be marked muscular atrophy. Scoliosis sometimes occurs and a form of club-foot with characteristic overextension of the great toe. In some cases a Babinski reflex has been seen. The fulgurant pains and crises of true tabes are not seen, nor is there paralysis of the bladder. Especially characteristic are the nystagmus and speech defects. The former is usually of the lateral variety. The speech is slow, labored, staccato, or scanning. Optic atrophy and ophthalmoplegias do not occur. The disease begins in childhood, as a rule, although in a few cases it does not appear until early adult life. It is an incurable affection, and steadily, but sometimes slowly, progressive.

Diagnosis.—From locomotor ataxia Friedreich's ataxia is distinguished by its early onset, its family association, its freedom from crises and bladder atony, its exemption from optic atrophy and ophthalmoplegias, its nystagmus, and its speech defects. There is a juvenile form of tabes, however, which may require care to distinguish it. It is due to hereditary (or very early acquired) syphilis, and presents genuine tabetic symptoms, such as pupillary changes, lancinating pains, and characteristic findings in the blood and cerebrospinal fluid.

Multiple sclerosis may simulate this disease, but it is rare in early childhood. It presents spastic paralysis with exaggerated knee-jerks, inten-

tion tremor, sometimes an optic atrophy, and it is not seen as a family affection.

In cerebral ataxia there may be exaggerated knee-jerks, optic atrophy, ataxic speech, and a more distinct cerebellar gait than in Friedreich's disease. Still these two affections have much in common.

The ataxic movements in this disease may simulate chorea and various kinds of tremors; but the association with the other symptoms is usually enough to prevent error. These movements have not the rhythm of tremors,

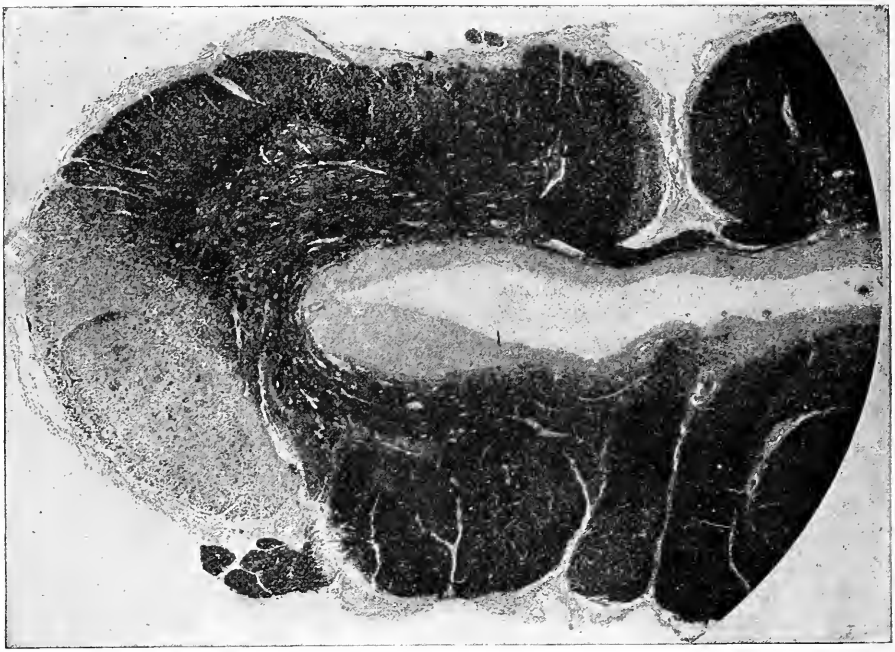


Fig. 393.—Syringomyelia in the cervical region.—Lloyd.

and they are not of the involuntary nature of choreic jerkings. In chorea the movements persist while the patient is at rest; in Friedreich's disease they are seen only during voluntary motion.

For the differential diagnosis between Friedreich's ataxia and syringomyelia see the description of the latter disease.

XI. SYRINGOMYELIA.

This term is applied to a process in the cord which results in the formation of a cavity. This process is a gliomatosis, or proliferation of a gliomatous tissue which breaks down in the centre. It is not a mere dilatation of the central canal of the cord, for this canal may not be included in the cavity, as was first pointed out by Simon in 1875. Dilatation of the central canal may result from other conditions, and is then properly called hydromyelia.

Pathology.—The overgrowth of gliomatous tissue is usually most marked in the gray matter. When this tissue breaks down, the resulting cavity is of greater or less extent. It may extend across the cord almost symmetrically on either side; in other cases it tends to follow one or other horn of gray matter. The resulting injury to the structures of the cord may be extensive. The anterior horns are involved, the lateral tracts below the lesion are degenerated, and the posterior horns may be almost cut off from the rest of the cord; but the posterior columns may largely escape. The location of the central canal is usually marked by a collection of ependymal cells on the anterior border of the cavity. The walls of the cavity are sometimes lined with a sort of membrane formed from gliomatous tissue. The lesion may be largely located in the cervical region, the cavity extending upward and downward in various shapes and to various levels. In a few cases the dorsal cord is most involved; in others, the lumbar cord. The cord at the seat of lesion is often flattened and ribbon-like.

FIG. 394.—Syringomyelia, showing scoliosis, muscular atrophy of the shoulder, and arthropathy of the ankle.—Lloyd.

Symptoms.—There is degeneration of the anterior horns. If the cavity is in the cervical cord, there results extensive muscular atrophy of the shoulders and arms with a flaccid paralysis and fibrillary tremors. Occasionally, however, the type of paralysis in the wasted limb is spastic, and the reactions of degeneration are wanting. The degeneration of the lateral tracts causes a spastic paralysis of the lower limbs, with exaggerated knee-jerks. The paralysis in rare instances is hemiplegic, and in some cases ataxia has been seen. The sensory symptoms are most characteristic: they constitute the so-called *dissociation* syndrome. There is loss of the sense of heat, cold, and pain, with preservation of the tactile sense. The location of this syndrome depends upon the seat and extent of the lesion: it may be most marked on the trunk, but it is also seen on the extremities. It may also be more marked on one side, and it occurs in areas of various extent. Occasionally some areas of tactile anæsthesia are also found. Trophic lesions occur. Maculæ appear on the skin of the legs; the toe-nails are enlarged and thickened, with transverse ridges. In the type known as Morvan's disease there are painless destructive lesions of the fingers. Scoliosis is not uncommon; and occasionally an arthropathy of one or other joint is seen just as in locomotor ataxia. Among the rarer symptoms are paralysis of the muscles of respiration, bulbar and oculomotor symptoms, paralysis

of the vocal cords, nystagmus, painless fractures, and various skin eruptions, such as urticaria and pemphigus. Bladder symptoms are usually not present.

Diagnosis.—Syringomyelia is to be distinguished especially from progressive muscular atrophy and amyotrophic lateral sclerosis. To both diseases it bears a resemblance because of the muscular atrophy of the shoulders and arms and the spastic paralysis in the legs; but it differs from both in its dissociation sensory syndrome and in its trophic lesions. The disease known as pachymeningitis hypertrophica of the cervical cord sometimes bears a striking resemblance to syringomyelia, even in its sensory symptoms. There may be the same loss of sense for pain, heat, and cold, with preservation of tactile sense; muscular atrophy of the shoulders and arms; and spastic paralysis of the legs; but in the former disease there is often a history of trauma, and in some cases the stiffness and deformity following upon fracture of the vertebræ are seen.

Morvan's disease is a form of syringomyelia in which there are painless destructive whitlows of the fingers (the *panaris analgesique* of French writers). Morvan claimed that the disease is distinguished, however, from syringomyelia, by the predominance of trophic lesions and the loss of tactile sense; but there may be the same muscular atrophy, scoliosis, and even arthropathy, and the two affections are probably closely allied. This type so much resembles some forms of leprosy that Zambaco and others have even claimed that syringomyelia is a form of leprosy—a curious instance of confusing a resemblance with an identity. There is no central gliomatosis in leprosy, and there are many distinctions, too numerous to mention here, which are described under that disease. The anæsthetic form of leprosy is dependent on a neuritis, not a cord lesion.

Charcot pointed out that one of the trophic lesions of syringomyelia is an enlargement of the hand closely resembling acromegalia. In his case the change was limited to one hand, and was symptomatic of gliomatosis. In some cases the paralysis is largely unilateral, resembling hemiplegia of cerebral origin; but the other cord symptoms distinguish it.

Dejerine contends that Friedreich's ataxia is due to a gliomatous change in the cord; and in a series of 12 autopsies in Griffith's collection of cases cavities were found in three. Doubtless in rare cases in which the posterior columns are much involved and ataxia results, the resemblance of syringomyelia to hereditary ataxia is manifest, but the sensory and trophic lesions of the one, and the speech defects and nystagmus of the other, serve to distinguish them. Thus in syringomyelia the knee-jerks are exaggerated, except in the rare tabetic form; in Friedreich's ataxia they are abolished, while in the latter there are speech defects which are not seen in the former, as well as a familial history in many cases. Nystagmus is most rare in syringomyelia, while it is common in Friedreich's disease. Finally, the dissociation symptom, muscular atrophy, arthropathies and other trophic lesions are all suggestive of syringomyelia; which disease, moreover, rarely begins in early youth.

In the rare cases in which ataxia occurs, especially if there should be a spinal arthropathy and lost knee-jerks, the resemblance to locomotor

ataxia may be striking, but the sensory symptoms, the muscular atrophy, the scoliosis, and the other trophic lesions would establish the diagnosis. The knee-jerks are not always lost in the ataxic form.

XII. SYPHILIS OF THE SPINAL CORD AND MEMBRANES.

Syphilis may confine its ravages entirely to the spinal cord and membranes, or it may affect both the spinal contents and the brain. The diffused cerebrospinal syphilis is usually more conspicuous for its brain symptoms, but evidences of involvement of the cord can often be found on close inspection.

Pathology.—Syphilis of the nervous system is due to the action of the *Spirochæta pallida*, but the exact mode of attack is still the subject of debate. Among early or initial lesions are lymphangitis, with exudation of lymphocytes in the perivascular spaces; meningitis of an exudative or gummatous type; and thickening of the walls of the arteries. The nervous tissue is involved secondarily, as is shown by sclerosis, system degenerations, and in some cases by necrosis or softening. It is highly characteristic of the syphilitic inflammation to become exudative; there then results a thickening of the membranes, a plastic exudate, and very often a gummatous neoplasm. In some forms the resulting meningomyelitis presents but little exudation and practically no gumma to the naked eye, but under the microscope the characteristic lymphocyte infiltration may be found. Thickening and even obstruction of blood-vessels may occur, leading to various degrees of necrosis or softening. In the spinal cord the lesion may be largely confined to the membranes, especially the pia, with only a limited area of peripheral myelitis underneath. This condition is especially seen about the lateral or posterolateral columns. In other cases the disease process is more diffused, without reference to the various tracts and systems of the cord, thus causing various bizarre combinations, and symptoms of disseminated myelitis.

Symptoms.—Because of its irregular distribution and various degrees, the syphilitic process gives rise to manifold and irregular symptoms. In some cases there is merely a meningomyelitis, more or less circumscribed, with some resulting sclerosis. The symptoms are then practically the same as have already been described under the head of myelitis. In the more diffused or disseminated form, the symptoms are irregular. Irritation of the nerve-roots is common, with resulting pains, girdle sense, and stiff back. There may be spastic paraplegia, impairment of the nerve supply to the bladder, and various forms of anæsthesia. Sometimes there is muscular atrophy, and even monoplegia. As a rule, spinal syphilis does not cause a so-called "system-disease." Exceptions to this rule occur, however, especially in the form described by Erb and sometimes called by his name. In Erb's paralysis the lesion is a meningomyelitis of the lateral aspects of the cord. There results a spastic paralysis of the legs, with exaggerated knee-jerks, low muscle tension, that is, without contractures; with weakness of the bladder, and usually no involvement of sensation. In some cases, however, the lesion includes the posterior aspects of the cord, and there is

then added an ataxia, possibly with some alterations of sensation. This condition is known as ataxic paraplegia.

An *essential* lateral sclerosis, in which the pyramidal or motor tracts are degenerated, very much as the posterior tracts are affected in locomotor ataxia, has been described by Lloyd and Ludlum.

Another form of spinal syphilis is seen in the gummatous tumor. This may be located at almost any level of the cord, or sometimes in the cauda equina. Its symptomatology will depend upon its location and extent, the same as in any other tumor of the spinal cord.

There is an acute form of syphilis of the cord, in which the onset is abrupt and the case progresses rapidly until it presents all the features of a transverse lesion, such as paraplegia, paralysis of the bladder and bowel, bed-sores, etc. In these cases softening and formation of cavities occur from occlusion of blood-vessels.

The clinical picture of spinal syphilis varies; there may be recessions and improvement; at other times, an irregular advance with remissions. Pure "system diseases" are rare, except in Erb's type, and the affection may especially change under specific treatment. In some cases there may be cerebral symptoms, and involvement of one or more cranial nerves. Pupillary changes are then seen, especially abolition of the reflex to light.

Syphilis is now known to be the sole cause of locomotor ataxia. The lesion in tabes, however, does not always present the type of an exudative meningomyelitis, but it is rather a parenchymatous change, a dystrophy confined to the sensory protoneuron. Nevertheless there are some pathologists, as Nageotte and others, who believe that even in locomotor ataxia the initial lesion is a meningitis affecting the posterior nerve-roots. However that may be, tabes dorsalis is usually described as a distinct disease.

Diagnosis.—Syphilis is likely to cause an irregular distribution of symptoms, but this fact is not of as much diagnostic importance as some writers contend. There are cases of myelitis, or meningomyelitis, with or without softening, in which it is not easy to make a differential diagnosis at the bedside. The problem is simplified in cases in which there is a clear history of syphilitic infection. In doubtful cases the Wassermann and other laboratory tests may be determinative. In cases in which there is some associated cerebral syphilis the diagnosis is more evident. It is worth while to bear in mind that syphilis is much the most frequent cause of meningomyelitis, and this fact raises a presumption in favor of its presence in any given case.

The same difficulty occurs in cases of Erb's palsy, for this affection closely resembles essential lateral sclerosis (the so-called primary spastic paraplegia), except that in the former the bladder is often involved, slight sensory changes occur, and the muscle tone is less increased. In Erb's palsy there may also be irritative symptoms, as pain and the girdle sense, due to involvement of the posterior nerve-roots. This is particularly so in cases in which the lesion spreads to and upon the posterior columns. We then see a condition of ataxic paraplegia, which is practically indistinguishable from the combined sclerosis which some authors describe as

a system disease of non-syphilitic origin. These fine problems in diagnosis cannot always be satisfactorily solved, but in all such cases the history of syphilis in the patient should be carefully sought; and whether this is found or not, the antisyphilitic treatment should be given a fair trial. The laboratory tests of the blood and cerebrospinal fluid will throw light on these cases. In recent years, as our knowledge of nerve syphilis has increased, it has become more and more evident that most of these clinical forms are mere variants of syphilis of the cord.

Some authors attempt to make a distinction between locomotor ataxia and syphilitic leptomeningitis extending to the posterior columns. In the latter disease there are sensory symptoms and ataxia closely resembling these symptoms in tabes, but the other distinctive tabetic symptoms, such as optic atrophy, Argyll-Robertson pupil, crises, and arthropathies, are wanting.

XIII. TUMORS OF THE SPINAL CORD.

We include here not only tumors of the cord proper but also tumors of the membranes, for the distinction between them is not clinically possible. We also include tumors of the vertebræ, for although these are sometimes distinguishable at the bedside from intraspinal growths, their clinical features are similar. Tumors of the spinal cord proper are rare; those springing from the membranes are the more common. In 50 cases collected by Mills and Lloyd, the largest number were sarcomata, gliomata, or gummata. In nearly one-half of these cases the tumor was in the cervical region; the dorsal region was involved next in frequency, and then the lumbar region. The cauda equina was also involved in a few cases.

Symptoms.— In most cases the symptoms indicate irritation early of the nerve-roots and membranes, and later pressure on the cord. Hence pain is often an early or initial symptom; it may be intense, neuralgic, persisting for a long time in one region, and may radiate or be located far from the cord (eccentric pain). It may be associated with hyperæsthesia, hyperalgesia, paræsthesia, or even anæsthesia, and there may be a subjective sense of numbness in some localized part, and even a girdle sense. With the pain there may be some stiffness of the spine, and contracture of some or other muscle groups. As the case progresses the symptoms of pressure show themselves; there is paralysis of one or other limb or group of muscles, and anæsthesia is more marked and more extensive. There may be anæsthesia dolorosa, that is, absence of sensation to objective tests, with the presence of pain in the affected part. Paralysis of the bladder and bowel may eventually come on. In fact, we see the symptoms of either a partially transverse or even (in advanced cases) a totally transverse lesion. The distribution of these symptoms will, of course, vary according to the seat of the lesion. In a very advanced stage there will be total paralysis below the seat of lesion, contractures, deviation of the spine, anæsthesia, incontinence, bed-sores, cystitis, alteration in the reflexes, dyspnœa, tachycardia or bradycardia, in cervical cases dysphagia, and even bulbar and ophthalmic symptoms. In some cases there is pain on pressure

or palpation over the site of the tumor, and pain on twisting or bending the spine.

An acute onset of paraplegia, with abolition of the knee-jerks, in a case in which there had only been a slight and obscure girdle sense for a few weeks, has recently been observed by the writer in the Philadelphia General Hospital. The tumor was in the membranes of the upper dorsal cord and proved to be a gumma.

Diagnosis.—The mode of onset is often suggestive. The initial symptoms are likely to be irritative, hence pain. This pain may be eccentric and localized in the distribution of one or of a few nerves. Paresis begins also as a localized symptom; it may at first be more marked in one limb or even in one group of muscles. Later the symptoms are more suggestive of compression of the cord, as anæsthesia, paraplegia, incontinence, and bed-sores. In some cases the symptoms are markedly unilateral, causing the so-called Brown-Séquard paralysis, or an approach to it.

From syphilis of the cord it is not always possible to distinguish tumor. In fact syphilis may cause a gummatous tumor. The mode of onset is more suggestive of a neoplasm. The Wassermann test and the other laboratory tests of the cerebrospinal fluid for syphilis should always be made. The progress of the disease in tumor is rather more persistent and hopeless than in most cases of syphilitic meningomyelitis. Recently a condition of xanthochromia of the cerebrospinal fluid has been described as indicative of a total transverse lesion of the cord: the fluid presents a yellow tinge, probably due to the retention of the coloring matter of the blood.

From myelitis and acute softening the distinction is often to be made by the more abrupt onset of these affections. The symptoms of a transverse lesion are much sooner established than in tumor; and the initial pain, so marked in case of neoplasm, may be wanting. Unilateral symptoms are not common in myelitis and softening. In hemorrhage the onset is sudden, often caused by trauma, and the disease reaches its acme in a short time. In spinal caries the bone lesion can usually be detected by inspection. The X-ray may demonstrate the lesion. In very early cases, before deformity appears, it may be possible to elicit pain by jarring the spine, and there may be stiffness of the back, and pain on passive movements of the trunk.

In traumatism the case can usually be distinguished by the history. Still, it is well to recall that tumor may follow trauma. Aneurism of the aorta may erode the spine and cause symptoms, first of irritation and later of pressure. It is only to be detected by exploration and by the methods of physical diagnosis. Cancer of the vertebræ usually causes very urgent symptoms, especially of initial pain, and later of pressure on the cord or nerve-roots. The nature of the lesion may remain for some time obscure; or there may be the history of precedent cancer. The symptoms soon become extremely aggravated, and the patient becomes cachectic.

In neuritis there is soreness of the nerve-trunks, with paralysis of motion or of sensation, or of both, in areas supplied by the individual nerves. In early stages motion is more likely to be impaired than sensation. Muscular atrophy occurs. Compression symptoms, and involvement of the

bladder and rectum, are wanting. Still, the distinction is not always easy between neuritis and intraspinal tumor, especially if the new growth affects chiefly the nerve-roots, as in the cauda equina.

The eccentric or localized pain of tumor, especially in the early stage, may closely simulate a mere neuralgia, or even a disease of some internal organ, as in the chest or abdomen. The differential diagnosis must depend on the association of the symptoms; for instance, of other symptoms of cord-lesion, on the one hand, or of disease of the suspected viscus, on the other.

The local or regional diagnosis is to be made as in cases of myelitis or trauma.

XIV. INJURIES TO THE SPINAL CORD.

The spinal cord is subject to injury by blows, falls, crushings, stab-wounds, and gun-shot wounds. The commonest injuries are those which also cause fracture and dislocation of the vertebræ. It is not essential, however, that there should be a fracture or dislocation of vertebræ, as fatal injury has been done to the cord by falls, without visible injury to the bones. Gun-shot wounds are common, and a few instances are on record of the cord being injured or partly severed by a stab with a knife or stiletto. The commonest seat of injury to the cord is in the neck: next in the dorsal region. The lumbar region, being more massive and better protected by large muscles, is not so often involved. It has been supposed in some cases that a vertebra may be partly dislocated and then spring back into place, thus causing a crushing of the cord. Fracture is not always associated with dislocation, nor *vice versa*.

The lesion in most of these cases is severe. The cord is either partly disintegrated, or entirely so in its transverse diameter. It is softened and necrosed, and may be the seat of hemorrhage. There may also be hemorrhage within the spinal membranes. In long-standing cases in which there have been attempts at repair, there is much scar tissue, together with degeneration of various tracts in the cord.

Symptoms.—The symptoms depend upon the seat of the lesion.

Injury to the *cervical region* causes a characteristic symptom-group, which varies according to the extent of the injury. There is a spastic paralysis of the legs, more or less complete, with paralysis of the bladder and rectum. The knee-jerks are, as a rule, increased, although in totally transverse lesions the knee-jerks may be abolished at first, according to Bastian's law. Later, if the patient survives, they may return and become exaggerated. This is also true of the other deep reflexes, but this is not an invariable rule, for the writer has seen a case of gun-shot injury to the cervical cord, causing complete paraplegia, in which the patient survived for months and the knee-jerks were not restored, although there was slight reaction towards the end. Contractures of the leg muscles often supervene, and bed-sores may form very rapidly about the buttocks or on the sacrum. The arms are totally or only partly paralyzed, according as the cervical enlargement and the roots of the brachial plexus are or are not totally involved. In some cases the arms lie paralyzed and flaccid at the side;

in other cases some power is retained, especially power of flexion of the arm at the elbow, and contractures supervene. Wasting of the muscles of the shoulders, arms, and hands is likely to set in if the cervical enlargement is injured. Sensation may be entirely abolished below the line of injury. If the lesion is low in the neck the anæsthesia may not involve the shoulders and outer aspects of the upper arms. Pain on moving the neck may be severe. If the injury extends above the fourth cervical segment the phrenic nerve may be paralyzed and cause death. The pupillary centre in the cord may be injured, with consequent contraction of the pupil; if it is only irritated the pupil is dilated. In some cases of injury to the cervical cord the clinical picture closely resembles syringomyelia. There is atrophic paralysis of the shoulders, arms and hands, spastic paralysis of the legs, with the dissociation sensory symptoms as seen in syringomyelia. In such cases there is usually deformity of the cervical spine from the old injury, and subsequent ankylosis.

Fig. 395.—Trauma of the cervical region of the spinal cord, simulating syringomyelia.—Lloyd.

Injury to the *dorsal region* causes spastic paraplegia and all the other symptoms as described above except those in the upper extremities. The anæsthesia when present gives a valuable clue to the uppermost limit of the injury. There may be a zone of hyperæsthesia at the extreme upper limit, due to irritation of the nerve-roots; and pain may radiate through the trunk at this level for the same reason.

Injury to the *lumbar region* also causes paralysis of the legs, bladder, and rectum, but if the lumbar enlargement is involved the paralysis of the legs is flaccid, with abolished knee-jerks, wasting of the muscles, and electrical changes. Anæsthesia may be present on the buttocks, genitalia, perineum, thighs, and legs. Pain in the legs may also be present.

A unilateral lesion of the cord may cause the so-called Brown-Séquard syndrome: there is paralysis of motion on the side of the lesion and loss of sensation on the opposite side, but this sensory loss is in the temperature and pain sense rather than in the tactile sense. If, however, the posterior column of one side is affected there is tactile anæsthesia also, but it is on the side of the lesion.

Diagnosis.—To determine the exact seat of the lesion the practitioner should study the uppermost limits of the anæsthesia, and the muscle-groups involved, and compare them with a chart and table of the spinal segments. In this way an exact local diagnosis may be reached (pp. 327, 328, Vol. I).

The history of the case is usually sufficient to establish the clinical diagnosis. It is not always possible to say whether the lesion is a mere hemorrhage in the substance of the cord, or in the membranes, or whether it is a crush of the cord. Practically the distinction is not of much importance so far as the welfare of the patient is concerned. It is more important to ascertain, if possible, whether the cord is merely affected by pressure, or whether it is actually crushed, especially when surgical intervention is contemplated. But this is seldom possible before operation. The reason why laminectomy is so seldom beneficial in these cases is that the cord has been damaged beyond the power of surgery to repair. Clinically these cases closely resemble myelitis; and in some of them secondary inflammation may be present. Abolition of the knee-jerks may indicate a total transverse lesion and is usually of grave import.

XV. HEMORRHAGE IN THE SPINAL CORD AND MEMBRANES.

Hemorrhage in the spinal cord, or hæmatomyelia, is usually the result of injury. It may be associated with a crush of the cord due to violence, or with fracture or dislocation of the vertebræ. In a few cases of injury, however, the hemorrhage may be the only lesion. Thus cases have been reported of great violence, as a fall down a long flight of stairs, in which the bones were uninjured, and even the membranes escaped, and yet a small and rapidly fatal hemorrhage was found in the substance of the cord. The hemorrhage may be in the gray matter, or close to it, and may cause extensive destruction of the spinal medulla. It may be so small as not to be apparent until the cord is sectioned, or it may cause an appearance of pallor or slight swelling on the surface. In some cases the blood breaks through to the surface and is present in the meninges. Minute capillary hemorrhages may be the starting-point for necrotic soft-

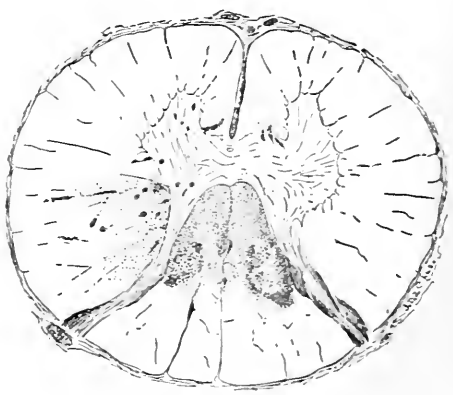


FIG. 396.—Hæmatomyelia.—Lloyd.

ening. Van Gieson has found long slender columns of necrosis in the cord, causing narrow cavities, which he thinks may be due to traumatic hemorrhage. They extend for long distances both above and below the main lesion. Meningeal hemorrhage may also occur from trauma.

Spontaneous, or non-traumatic, hemorrhage in the spinal cord is rare. The primary lesion is probably vascular, due to some weakness or disease of the blood-vessels. It is remarkable that such vascular lesions, which are so common in the brain, are apparently so uncommon in the spinal cord. It has been doubted by competent pathologists whether arteriosclerosis, as well as thrombosis and embolism, occurs in the cord; but there is not sufficient ground for dogmatic statements in the negative. It seems more probable that some cases of hemorrhage and hemorrhagic softening in the cord may be due to such lesions.

Symptoms.—The symptoms come on rapidly, even suddenly, and may cause profound shock; but even in the traumatic cases this rule is not absolute, for hours may elapse before all the symptoms are established. This is probably due to the fact that a small hemorrhage, once started, continues for some time to progress, with gradually increasing effect. Especially in meningeal hemorrhage this gradual onset is seen, and perhaps more so in the lower part of the spinal canal, where the cauda equina is involved. Many hours have been known to elapse before the paralysis was complete. In such a case presumably the blood gradually settles and clots in the lower part of the spinal canal. There may be complete paralysis of all the muscles below the level of the lesion. If the lesion is in the neck, the muscles of the chest may be paralyzed. The respiration is then diaphragmatic and irregular.

All modes of sensation may be absolutely lost below the level of the lesion. These symptoms, however, vary. Some hours may elapse before they are fully established, and later, even in unfavorable cases, sensation has been known to return in part. Thus the patient may have a return of tactile sense in some limited area; or of pain sense, or thermic sense, or sense of position, one or all. The bladder and rectum may be paralyzed, and priapism may occur.

The reflexes, especially the knee-jerks, may be retained and soon become exaggerated. If the lesion is totally transverse, the knee-jerks are likely to be abolished, although they may return in time. Occasionally they are not lost until after some days. Total abolition of the knee-jerks, therefore, in a hemorrhage *above* the lumbar enlargement is not a favorable sign, for it indicates an extensive lesion. If the hemorrhage is *in* the lumbar enlargement the knee-jerks will probably also be abolished, but this is from interference with the reflex centres in the cord; hence it is not necessarily so ominous a symptom. The ciliary reflex from irritation of the skin of the neck may be absent in cases of hemorrhage in the cervical region; hence there may be spastic myosis. If this centre is only irritated there is symptomatic mydriasis in one or both eyes.

Pain may be an urgent symptom. In Kindred's case, in which the hemorrhage was in the fourth dorsal segment, there was an initial agonizing pain simulating angina pectoris. Pain may persist, and it may even be felt far below the level of the lesion, if this is not totally transverse; this probably

because free blood escapes in the meninges and causes pressure and irritation at lower levels. The girdle or "cincture" feeling is present in some cases.

In brief, the symptoms of hæmatomyelia, whether traumatic or non-traumatic, are those of a complete or almost complete transverse lesion of the cord. Abolition of the knee-jerks may be seen. The localizing symptoms depend upon the exact level at which the lesion occurs, and they have already been described under the head of myelitis.

The course of severe cases is usually rapid. In Kindred's case of spontaneous hemorrhage death came in six hours. If the lesion is totally transverse, with abolished knee-jerks and rapidly forming bed-sores, the prognosis is highly unfavorable. If the lesion is high in the neck death may come from paralysis of the phrenic nerve.

High temperature and excessive sweating are seen toward the end in fatal cases. In a case recorded by Lloyd moist skin was noted above, and dry skin below, the line of anæsthesia toward the end.

In hemorrhage in the membranes, when the cord itself is not involved and not much pressed upon, the outlook is more favorable, although permanent crippling in some form is not unusual. Pain is often a prominent symptom, and the anæsthesia and pain may follow individual nerve-trunks. In the legs, if the cauda equina is involved, there is a flaccid paralysis with muscular atrophy and abolished reflexes, somewhat like a multiple neuritis, but with paralysis of the bladder and bowel.

Diagnosis.—In traumatic cases the diagnosis is not difficult, except to distinguish hemorrhage from crush, and this is often not possible, nor is it of great practical importance. The history of the case points unerringly to a grave lesion of the cord. It is important to distinguish fracture, and this can be done only by a careful surgical examination, aided by the X-rays. Hæmatomyelia is not likely to be mistaken for a disease of any internal organ, and yet in dorsal hemorrhage the initial symptoms have simulated angina pectoris. But the speedy onset of paralysis, anæsthesia, priapism, and incontinence would distinguish the cord lesion. So, too, of pain from lumbar hemorrhage simulating disease of abdominal viscera. The supreme difficulty in non-traumatic cases is to distinguish hæmatomyelia from transverse myelitis, acute white softening, and pachymeningitis. The sudden onset and rapid course serve better than all other symptoms to distinguish hemorrhage from any of these conditions; but the diagnosis in non-traumatic cases may remain obscure. The extreme rarity of spontaneous hæmatomyelia must not be overlooked in coming to a conclusion. Softening and hemorrhage being due often to the same causes, and being part of the same pathological process, a dogmatic diagnosis between them may sometimes not be practicable. A lumbar puncture may show the presence of blood in the cerebrospinal fluid. In acute spinal syphilis, with abrupt onset of symptoms, the tests for the Wassermann reaction and for globulin (Noguchi and Nonne), and the presence of a lymphocytosis, might be determinative. The exact seat, or level, of the lesion is to be determined as in injury or myelitis.

XVI. SOFTENING OF THE SPINAL CORD.

Acute softening of the cord is often confused with myelitis, and indeed in some cases it may be impossible to distinguish between them at the

bedside. The subject has already been discussed under the head of myelitis. The latter term properly indicates an inflammation, and this in turn is due to some infection; whereas softening may presumably be caused by a vascular lesion, such as embolus or thrombus. Nevertheless, softening is sometimes secondary to inflammation; and it also results from trauma. Syphilis, causing a meningomyelitis and endarteritis, may also cause softening. Weiss has recorded a case of softening of the cord occurring suddenly in a boy who had mitral disease. Gowers refers to a similar case. The inference is that cardiac emboli were the causes of the softening. The symptoms are so similar to those which occur in myelitis that it is not necessary to describe them again. The onset of the disease, however, may be more abrupt.

XVII. THE CAISSON DISEASE.

The caisson is a large compartment, inverted, in which a constant supply of compressed air is maintained in order to counteract the pressure of the water from without, thus allowing men to work upon the foundations of piers. The caisson disease is an affection, largely of the central nervous system, and especially of the spinal cord, caused by working in the compressed air of these compartments. The highest pressure attained at the St. Louis bridge was 50 lbs. to the square inch, the normal pressure of the atmosphere being 15 lbs. By some observers the mischief is attributed to the emerging from the compressed air while the system is overheated and fatigued with the hard labor.

Pathology.—Van Rensselaer studied the cord in a case of this disease and found extensive changes. A disseminated necrotic area was found in the dorsal region, with ascending degeneration in the columns of Goll and in the direct cerebellar tract, and descending degeneration in the pyramidal tracts, respectively above and below the mid-dorsal lesion. The necrosis seemed to be confined to the white substance, the gray matter not being involved. Possibly with the more refined methods now used, this entire exemption of the cells of the gray matter might not be found. No hemorrhages were seen. The theory that air-bubbles or air-emboli cause the softening has been advanced. Brooks has recently studied the blood-pressure in 75 workmen before, during, and after working in the caisson, and found no marked change in arterial pressure.

Symptoms.—The initial symptoms do not appear until after the workman emerges into the outer air. Some minutes, even hours, may elapse. There is usually severe epigastric pain, with vomiting, then very severe pain in the back and lumbar region, along the spine, and shooting down the legs. Smith described these pains as of a tearing character and intolerable. Anæsthesia shows itself promptly, often of the type known as "anæsthesia dolorosa," and advances with the motor symptoms. The paralysis is usually in the form of a paraplegia, although in some cases the arms also have been involved. The bladder and rectum are commonly paralyzed. Bed-sores form.

Brain symptoms are seen in the more severe cases, and especially in fatal cases. There are headache and vertigo, and in fatal cases unconsciousness supervenes before death. Pelton describes a comatose type,

which may appear suddenly, with cyanosis, and is usually fatal. The duration in mild cases is from a few hours to six or eight days. In severe cases death may result in a few days.

The viscera are not, as a rule, seriously involved. There may be bronchial irritation and cough. The urine has usually a high specific gravity. Perforation of the ear drum and catarrh of the middle ear have been observed.

Diagnosis. — The import of severe spinal symptoms, such as paraplegia, anæsthesia, and incontinence, in a caisson worker soon after quitting work is unmistakable. The disease, as described by most observers, is of spinal or central origin, as proved also by such autopsies as the one made by Van Rensselaer, and is not a mere peripheral neuritis, due to carbonic acid poisoning, as others have contended. Still, it is not unlikely that the peripheral neurons may be found involved in some cases. An affection similar in every way to the caisson disease occurs in divers.

XVIII. AFFECTIONS OF THE CAUDA EQUINA.

The cauda equina is the leash of nerve-roots lying in the lower end of the spinal canal. These are the nerve-roots that come off from the lumbar and sacral segments. The spinal cord ends about opposite the second lumbar vertebra; consequently the nerve-roots from its lower segments have to traverse comparatively long distances before uniting to form the lower spinal nerves at or about their respective foramina of exit from the spinal canal. These prolonged nerve-roots are the cauda equina.

Pathology.—The cauda equina may be the seat of injury or disease. The injuries are similar to those that cause damage to the cord itself. Thus there may be fracture or dislocation of the vertebræ, rupture of the membranes, or traumatic hemorrhage, sometimes with secondary inflammation. Fractures and dislocations of the lower parts of the spine are not so common as in the dorsal and cervical regions, for the parts are massive and protected by large muscles; nevertheless they sometimes occur. But a traumatic meningeal hemorrhage may settle from still higher levels and press upon the strands of the cauda equina. Septic meningitis may occur in the lower part of the spinal canal, sometimes during the puerperium, or from other causes, such as a sloughing bedsore. Syphilitic meningitis may also occur, and syphilitic tumor, or gumma, is not so very uncommon. Other varieties of tumor may also be seen. It is well to bear in mind that although the cauda equina, being composed of nerve-fibres, is anatomically a part of the peripheral nervous system, nevertheless, being contained within the spinal canal and enclosed within the spinal meninges, it has practically some points of resemblance to central structures.

Symptoms.—Pain is a common symptom in meningitis or neuritis of the cauda equina, and is due to involvement of the sensory roots. It may be felt in the lower part of the abdomen or pelvis, or it may be transmitted to the legs and feet. It is sometimes lancinating or neuralgic, or burning as in neuritis, and may be associated with glossy skin

and cutaneous hyperæsthesia. Anæsthesia may also occur; it may be variously distributed, and is sometimes segmental in type, especially when the lesion is localized, as in the case of hemorrhage, and involves only the lower parts of the leash of nerves. This is explained by the fact that each succeeding pair of nerves from below upward represents a distinct segment of the cord.

The paralysis is peripheral in type; that is, it is of the flaccid variety, with atrophy of the muscles and changes in the electrical reactions. The knee-jerks are abolished, unless the roots from the upper lumbar segments escape. This type of paralysis is due to the fact that the strands of the cauda equina belong anatomically to the peripheral nervous system.

In a few cases the knee-jerks are exaggerated, as when the lesion extends gradually from below upward and acts as an irritant to the roots from the upper lumbar segments. The distribution of the paralysis varies. In some cases muscular groups only are involved, as the peronei, sural, hamstring, etc., thus indicating a segmental type.

The bladder and bowel may be paralyzed, and priapism or even normal erections do not occur as a rule. Bed-sores are not uncommon. In some cases painful contractures are present in the legs.

Tumors of the cauda equina cause a great variety of symptoms. All depends upon the level at which the tumor is located and its extent. Pain is an early symptom, possibly felt low in the back, in the pelvis, or in the legs. Later, pressure symptoms develop, and consist of various forms of paralysis, usually of the peripheral type. The symptoms thus gradually extend as from a centre. The bladder and bowel may be affected.

Diagnosis. — The diseases from which these affections have to be differentiated are myelitis, multiple neuritis, and locomotor ataxia. In traumatic cases the history is usually sufficient to distinguish them. From myelitis the peculiar segmental distribution of the symptoms, the flaccid atrophic paralysis, the reactions of degeneration, and the lost knee-jerks suffice to make clear the difference. It must be admitted, however, that in myelitis involving the lumbar enlargement all these symptoms might be present, and in such a case an exact diagnosis may not be practicable.

Inflammation of the cauda equina may closely simulate multiple neuritis, but multiple neuritis is seldom confined to the legs, and the bladder and bowel are, as a rule practically without exception, not involved. In the cases of alcoholic multiple neuritis in which the lower limbs alone are paralyzed, the exemption of the bladder and bowel would serve to point out the difference. The pains, too, are of a different kind in multiple neuritis, being more local and peripheral, and felt most acutely on pressure on the nerves and muscles. The Wassermann test should be made, as well as the other laboratory tests of the cerebrospinal fluid for syphilis. An excess of lymphocytes is characteristic of syphilis, and blood-cells would doubtless be found in cases of hemorrhage.

Disease of the cauda equina resembles locomotor ataxia only remotely, and chiefly in the pain and lost knee-jerks. There are no true tabetic symptoms, as ataxia, pupillary changes, and optic atrophy; and even the pain is not like the paroxysmal fulgurant pain of tabes. Moreover in tabes

we do not see distinct segmental anæsthesia, and the muscles, at least in the early stages, are not paralyzed and degenerated. Nevertheless, it is claimed by some observers that in the early stage of tabes the sensory changes, such as analgesia and hypæsthesia, suggest a nerve-root distribution.

Tumor of the cauda equina is discussed with tumors of the spinal cord.

XIX. SPINA BIFIDA.

Spina bifida is a developmental defect, caused by failure of the vertebral arches (which grow from the mesoblastic somites in the embryo) to coalesce behind the spinal cord and between it and the skin of the back. Several varieties of deformity result according to the tissues involved (see Fig. 10, Vol. II). In meningomyelocele the cord also is involved in the defect of development, but in simple meningocele the membranes alone are included in the tumor.

Spina bifida is sometimes associated with other developmental defects, especially hydrocephalus.

This condition usually presents no difficulty in diagnosis, although the defect is not always detected at once in the new-born child. The small tumor on the back tends to grow, and in time may become very large. The important point is to determine the variety of the bifid spine. In meningomyelocele the spinal cord and nerves are involved, and paraplegic symptoms are always present. This distinction is important, especially from a surgical standpoint, for any operation for the excision or obliteration of the sac in this variety, even if successful, must inevitably leave the child a more or less hopeless cripple. In simple meningocele the results may be better, as the cord is not involved. An X-ray picture may be made, but it may be doubted whether it could lead to a successful distinction between the types of spina bifida.

The tumor is usually in the lumbar, lumbosacral, or sacral region. It is infrequent in the cervical and upper dorsal regions.

Spina bifida requires to be distinguished from certain other embryological defects, such as congenital tumors in the sacrococcygeal region, dermoid cysts, and teratomata, but these fall within the purview of surgery.

DISEASES OF THE SPINAL NERVES.

I. MULTIPLE NEURITIS.

This disease, also called peripheral neuritis or polyneuritis, is, as its name indicates, an inflammation, more or less wide-spread, of the nerves.

Etiology.—Multiple neuritis is caused by a great variety of poisons, the most common being alcohol, lead, arsenic, mercury, and some of the infectious diseases, especially diphtheria. Cases also occur after typhoid fever, and more rarely after smallpox; also in diabetes. Beriberi is a form of multiple neuritis, the exact causation of which is still undetermined; a diet of polished rise has been suspected, but some kind of infection seems more probable. Among the rarer causes of polyneuritis are carbon monoxide and carbon bisulphide. Phosphorus may also cause neuritis, and in ergotism there is involvement of the sensory and motor nerves. The anæsthetic form of leprosy is due to a neuritis. Among other assigned causes are influenza, malaria, and emetine as used for amœbic dysentery.

Pathology.—There are two forms usually described—the paren-

chymatous and the interstitial. Whatever may be the cause of multiple neuritis, its essential morbid anatomy is much the same, and sometimes the cases partake of both forms. There are segmentation of the myelin, proliferation of the nuclei of the sheath of Schwann, and destruction of the axis-cylinder. In many cases there are also some overgrowth of the connective tissue and alterations in the blood-vessels. In the purely parenchymatous form there is destruction of the neuron, even including its cell-body; but according to Berkley, in experimental poisoning with alcohol, the earliest changes appear to be in the blood-vessels of the nervous system. The seat of the most active changes is usually in the periphery of the nerves. As the cord is approached the disease process diminishes. At the present time, however, the tendency is to find some evidence of degeneration even in the cord, due doubtless to the peripheral neurons being implicated as far as their course in the spinal medulla—as, for instance, in the posterior columns.

Symptoms.—The most common form of multiple neuritis is that which is caused by alcohol. This may be taken as a type, although some of the other forms, notably that caused by diphtheria, differ from it in certain particulars, as will be pointed out.

Fig. 397.—Alcoholic multiple neuritis, showing wrist- and foot-drop.—Lloyd.

In *alcoholic neuritis* the earliest symptom is usually pain. This pain is felt especially in the nerve-trunks and in the muscular masses, such as the calves of the legs. It is commonly of an intense burning character, and is so urgent that for a while it may mask the other symptoms. When fully developed this pain is quite unmistakable; it is increased by pressure and by handling the parts; and the patient, especially if a woman, will cry out and even weep when the limbs, and particularly the legs, are handled. The toes and soles of the feet may be exquisitely sensitive, and sometimes exhibit some erythema. This is the state known as causalgia. The pain is increased on voluntary motion, and is usually worse in the legs and feet; but it may not be absent in the upper limbs. Pain is not present in all cases. Other sensory symptoms are paræsthesia and various grades of anæsthesia. The thermal sense may be affected in advanced cases, and the electrical sensibility may be impaired, especially to mild currents; but strong currents, particularly if they cause muscular contraction, are most painful.

Of motor symptoms the commonest are cramps, tremors, paralysis, contractures, and ataxia. Cramps occur early, but they are not seen in all cases. Tremors are occasionally seen in the weakened muscles, and are especially common in the alcoholic cases. Paralysis is the most common motor symptom. This is of the flaccid or peripheral type; the muscles waste and become flabby, and complete reactions of degeneration are seen. The distribution of this paralysis is characteristic; it invades all four

limbs, and is most marked in the extensor muscles; hence there are wrist-drop and foot-drop. It is also most marked in the distal muscles; that is, in the muscles furthest from the trunk; hence the forearms and hands, and legs and feet, are more paralyzed than the upper arms and thighs. The trunk muscles, however, do not escape, and the external muscles of respiration may be seriously involved. There is also tachycardia. The bladder and bowel are not paralyzed, as a rule, although a few doubtful cases are recorded. In the alcoholic cases the cranial nerves, except the vagus, are not often affected. Paralysis of the abducens nerve and optic neuritis have been observed. Patrick and others have seen facial diplegia in multiple neuritis, but this complication is not often, if ever, seen in the alcoholic cases.

FIG. 398.—Alcoholic polyneuritis, with marked muscular atrophy and wrist-drop.—Singleton Smith.

Muscular contractures are not uncommon in advanced cases; they are most marked in the flexor muscles, which are the least paralyzed. These contractures hold the limbs, especially the legs, in a semiflexed position; they are hard to overcome, and greatly retard recovery.

Ataxia is seen occasionally, giving rise to *pseudotabes*. The incoordination is not unlike that of locomotor ataxia. It occurs in the alcoholic cases and in those caused by lead.

The reflexes are usually abolished. This is true especially of the deep, or tendon, reflexes. The knee-jerks, as a rule, are lost early; in some cases, however, they may persist much diminished. Some observers claim that the deep reflexes are exaggerated in the early stage of the disease; but such exceptions are extremely rare, and they are difficult to explain except on the theory of irritation of the sensory neurons. The superficial, or skin, reflexes are not so promptly or uniformly lost, and in cases in which there is marked hyperæsthesia they may even be very active; but in advanced cases, particularly when there is anæsthesia, these reflexes are abolished.

Of trophic lesions the commonest is atrophy of the muscles. Œdema of the paralyzed legs is sometimes seen, and occasionally erythema, as of the soles of the feet. Ulcers and skin lesions are very uncommon; but trophic bed-sores, especially about the malleoli, have been observed in the post-typhoid cases. Glossy skin may be entirely absent. Profuse sweating is seen in some cases.

In severe cases the weakness of the heart muscle may be the determining cause of death. Paralysis of the external respiratory muscles constitutes an additional source of danger. The phrenic nerve is not often involved.

In some of the alcoholic cases a characteristic psychosis occurs. It is marked by a wandering delirium, with hallucinations of sight and hearing, confusion of identity of time, place, and persons, and a tendency to fabulation. Atypical cases of alcoholic multiple neuritis occur. Occasion-

ally, the patient has little if any pain, while the motor paralysis may be extreme. This *motor type* may or may not be associated with great ataxia, and constitutes a form of *pseudotabes*. It does not follow, however, that in all cases of pseudotabes there is an absence of pain. Another rather rare form is that in which the paralysis is confined to the lower limbs. The usual pain and sore muscles in the calves are present, but there is an absence of bladder and rectal troubles, such as occur in cord lesions. The knee-jerks are lost.

In *lead neuritis* the clinical picture is somewhat different. The distribution of the paralysis is usually much less extensive; as, for instance, in the cases in which the extensors of the forearms are alone involved. In some rare cases, however, there is a more wide-spread paralysis, and the upper arms, shoulders, and even the lower limbs are affected. There is a form of lead palsy closely resembling progressive muscular atrophy. The pseudotabes may also occur, but as a rule sensation is not much involved in lead cases, and pain is rarely observed.

Fig. 399.—Peripheral neuritis, with foot-drop of right side, after enteric fever.—Pennsylvania Hospital.

Postdiphtheritic paralysis (*q.v.*—diphtheria).

The two most specialized forms of multiple neuritis are *beriberi* and the *anæsthetic variety of leprosy* (*q.v.*—beriberi; leprosy).

In *diabetes mellitus* neuralgic pains of a severe type sometimes occur, and occasionally anæsthesia, especially of the legs, and along with this may occur some paralysis and atrophy of the muscles, with lost knee-jerks.

The forms of multiple neuritis due to arsenic, mercury, typhoid fever, smallpox, and most other infectious diseases show nothing very distinctive. The cases following typhoid fever were probably more frequent when the practice prevailed of giving large doses of alcohol in that disease.

Diagnosis.—The disease with which multiple neuritis is most likely to be confused is *locomotor ataxia*, and this is true especially of the ataxic type of polyneuritis. But polyneuritis differs from tabes in its mode of onset, which is usually much more brusque; in its history, which usually points to the poison or infection which causes it; in the pain, which is constant, burning, and neuralgic, and much increased by pressure, while in tabes the pains are lacinating, paroxysmal, and not affected by pressure, unless sometimes, indeed, they are relieved by it; in the paralysis and muscular atrophy, which are late phenomena in tabes and often absent even late in the disease; in the reactions of degeneration; in the gait, which is high-stepping in neuritis, with foot-drop, the toe grazing the ground (the

so-called "turkey-gobbler walk"), due to paralysis of the extensor muscles, while in tabes it is incoördinate, the foot being flung far out and the heel striking the ground first—a distinction which is seen even in pseudotabes of neuritic origin. Moreover, in locomotor ataxia there are the true tabetic phenomena, such as optic atrophy, Argyll-Robertson pupil, atony of the bladder, sexual impotence, and arthropathies, which are not seen in polyneuritis, although in the form due to lead, optic neuritis and atrophy sometimes occur. Some recent writers claim that the Argyll-Robertson pupil is seen occasionally in chronic alcoholism; but it must be very rare, and it always suggests a syphilitic infection. In both diseases the knee-jerks are abolished, and the loss of static equilibrium may be seen in the pseudotabes as well as in the true tabes. Sensory changes are somewhat similar in the two diseases, except that deep sensibility (the sense of position, of pressure, and of voluntary and passive motion) is more likely to be abolished in tabes, while the superficial sensibility (tactile sense especially) is preserved. But these modes of sensation are sometimes affected also in multiple neuritis.

Various forms of *myelitis* simulate multiple neuritis. Epidemic poliomyelitis, or infantile paralysis, usually begins abruptly with constitutional symptoms, the pain is of a rachialgic type, and the paralysis is likely to be more limited in extent than in multiple neuritis. A polyneuritic form of the disease, however, is sometimes seen, but the history is entirely different from that of multiple neuritis. In the subacute and chronic anterior poliomyelitis there is a flaccid paralysis with muscular atrophy, but pain is not conspicuous and may be entirely wanting, and fibrillation of the muscles is seen. Sometimes the tendon reflexes are not entirely lost. The reactions of degeneration are not complete; and finally the history and evolution are different. Transverse myelitis is marked by spastic paraplegia, with exaggerated knee-jerks, and incontinence. When the lumbar enlargement is involved there may be flaccid paralysis, but the bladder and bowel are paralyzed, and the symptoms are confined to the lower limbs.

In the *myopathies* the evolution of the disease is extremely slow; pain is not conspicuous, or it is even wanting, anæsthesia is usually absent, and the history of the case is different. The neuritic form of the myopathies has more in common with neuritis, and may even depend upon a slow neuritis, but the course is extremely slow, the disease is sometimes familial, and the history is different. Some of these forms begin in childhood, whereas multiple neuritis is usually, but not always, a disease of adult life. A few cases of alcoholic multiple neuritis have been reported in children.

Careless observers have mistaken the pain of multiple neuritis for *rheumatism*, and treated the patient accordingly. There is no inflammation, swelling, pain, or redness of the joints as in the latter disease.

Hysteria may simulate multiple neuritis, but only superficially, and more particularly in chronic cases, with anorexia and wasting and contractures of muscles. But there are no true reactions of degeneration; the tendon reflexes are preserved or even increased; hysterical stigmata are present; and the history is characteristic.

II. THE CERVICAL NERVES AND CERVICAL PLEXUS.

The cervical plexus is formed from the four upper cervical nerves. These nerves after issuing from the spinal canal divide into anterior and posterior branches; it is from the anterior branches alone that the cervical

plexus is formed. The posterior branches are largely sensory, and the largest nerve arising from these is the great occipital nerve. The most important branch of the plexus proper is the phrenic nerve.

Pathology.—This group of cervical nerves may be involved in diseases about the base of the skull and the upper cervical vertebræ. Among these especially are spinal caries, and, very rarely, new growths. Fracture and dislocation of these vertebræ occasionally occur. Pure neuralgic affections also may be seated in these nerve branches.

Symptoms.—Neuralgia of the great occipital nerve, which is distributed to the occiput as high as the vertex, is a distressing malady. There are painful points, especially where the nerve penetrates the trapezius muscle, and over the boss of the parietal bone. The pain is usually paroxysmal. This nerve may also be involved in dislocation of the atlas, as in a case reported by Lloyd, in which there was anæsthesia in the distribution of the great occipital (see Fig. 388). This is explained by the fact that the nerve is a branch of the second cervical nerve which issues from the spine between the atlas and the axis. In spinal caries and new growths there is not only pain but also stiffness of the neck muscles, and this may also be seen in meningitis in the posterior basic region of the brain. In some cases of torticollis it is probable that some of the motor branches of the plexus are involved.

Diagnosis.—Care is required especially to detect if possible any organic lesion, as new growths, spinal caries, fracture, basal meningitis, etc.

III. THE PHRENIC NERVE.

This nerve supplies the diaphragm. It arises from the third and fourth cervical nerves, and therefore has its central origin in the third and fourth cervical segments of the spinal cord. It receives a small branch also from the fifth cervical. In the neck it is deeply situated and passes between the subclavian artery and subclavian vein. It is supplied to the under surface of the diaphragm.

Pathology.—Owing to its deep situation in the neck and chest the phrenic nerve is not often injured. It may be involved in blows and wounds of the neck and in surgical operations; also in caries of the cervical spine, and in cervical meningitis. In injuries to the cervical cord, due to fracture or dislocation of the vertebræ, this nerve is sometimes involved in its centres of origin. This complication is then the immediate cause of death. In some of the cases in which the lesion is lower in the cervical cord, and the external respiratory muscles are paralyzed, life may hang upon the phrenic nerve, the respiration being entirely diaphragmatic. This nerve is said also to be involved sometimes in alcoholic multiple neuritis and in diphtheritic paralysis. It may also be pressed upon by tumors.

Symptoms.—Paralysis of both phrenic nerves causes complete paralysis of the diaphragm. Respiration is entirely by the external respiratory muscles. Dyspnœa may or may not be present. Bronchitis and pneumonia are among the risks in these cases.

Diagnosis.—The symptom may be overlooked, especially if only one phrenic nerve is affected. On close inspection, however, it is seen in the latter case that the movement of the diaphragm on the affected side is impaired, and when both nerves are paralyzed the failure of the diaphragm to descend is evident. Compensatory action of the external respiratory muscles causes exaggeration of the costal type of respiration.

IV. THE BRACHIAL PLEXUS.

The brachial plexus is formed from the anterior branches of the last four cervical nerves and of the first dorsal nerve. It begins close to the vertebræ and extends to just beneath the clavicle, where it gives origin to its main nerve-trunks. These trunks go to form the circumflex, musculospiral, musculocutaneous, median, ulnar, and internal cutaneous nerves. Hence the brachial plexus supplies with motor and sensory filaments the shoulder and upper limb. These various nerve-trunks, however, take origin from the brachial plexus in such a way that none of them is derived entirely from any one spinal nerve-root.

Pathology.—The brachial plexus may be the seat of severe neuralgia, trauma, inflammation, and neoplasm. Neuralgic affections are often of obscure nature; they may be caused by rheumatism or gout, possibly also by exposure and by slight grades of inflammation. Tumors are rare; aneurism sometimes acts as a cause. Trauma may cause extensive lesions. Fracture of the clavicle and dislocation of the head of the humerus, especially the subcoracoid form, may cause injury to the plexus. Spinal caries may cause irritation of the nerve-roots. There is a form of birth palsy caused by injury to the brachial plexus.

Symptoms. — In neuralgia usually the sensory fibres alone are involved. Painful points are present, as where the nerve-trunks are most exposed or most easily subjected to pressure; hence, on the side of the vertebræ, in the axilla, and on the musculospiral nerve in the upper arm, and on the ulnar and radial nerves in the forearm. If inflammation is present there may be areas of paræsthesia and anæsthesia, and vasomotor and trophic lesions. In these cases the motor fibres also suffer, paralysis resulting. The pain is sometimes intense.

In traumatic cases the pain may be very severe, and various forms of paralysis result according as the lesion is total or partial; also glossy skin and other trophic lesions, such as muscular atrophy; also contractures in old-standing cases. In organic lesions of the plexus from whatever cause there may be complete or almost complete brachial monoplegia. All voluntary movements are lost when the lesion is a total one, and the arm then hangs inert at the side. The shoulder may be kept slightly elevated by over-action of the trapezius muscle. The paralysis is flaccid, with reactions of degeneration, and the tendon-reflexes are lost. The pain may be severe, paroxysmal, ill-defined, and may interfere with sleep. In total lesion the anæsthesia involves the hand and forearm and the outer aspect of the upper arm. The tip of the shoulder is not involved, as it is supplied by the cervical plexus, and the inner aspect of the upper arm also escapes, as it is supplied by the first three dorsal nerves. The upper arm type, according to Erb, is caused by injury to the fifth and sixth cervical roots; the lower arm type by injury to the seventh and eighth cervical and dorsal roots. The upper arm type shows paralysis of the deltoid, biceps, brachialis anticus, supinator longus, and possibly the pectoralis major, infraspinatus, supraspinatus, and serratus magnus. The lower arm type is characterized by paralysis of the muscles of the lower arm and hand.

Diagnosis. — The diagnosis is easy, and is made from the peculiar distribution of the symptoms. Cases vary according to the extent and completeness of the lesion. The cause in pure brachial neuralgia may be obscure; and careful search should be made for organic lesions, such as neuromata, aneurism, and vertebral disease.

V. THE ANTERIOR THORACIC NERVES.

These nerves are not of great clinical importance except as pointers in some cases of injury to the brachial plexus. They are two in number. The external nerve is a branch from the outer cord of this plexus and supplies the pectoralis major muscle; the internal nerve arises from the inner cord and supplies both the pectoralis major and minor muscles. They are consequently likely to be involved in a lesion high in the plexus, but they escape in a lesion as low as the retroclavicular portion of that system of nerves. Paralysis of the two pectoral muscles impairs the use of the arm; but the incapacity to the patient is not as great as might be supposed. Forced adduction of the arm across the chest is affected.

There are a few cases on record of congenital absence of the two pectoral muscles on one side. Whether this anomaly depends on primary defect of the two nerves, is not determined. There is surprisingly little embarrassment to the patient, and in one case the individual was a laboring man who had found no inconvenience from his defect.

VI. THE POSTERIOR THORACIC NERVE.

The posterior, or long, thoracic nerve (the external respiratory nerve of Bell) arises from the fifth and sixth cervical nerves. It passes through the middle scalene muscle in the neck, and runs upon the outer surface of the serratus magnus muscle, which it supplies with a filament at each of its digitations. It supplies no other muscle.

Pathology. — This nerve is most exposed to injury at and about the point where it penetrates the middle scalenus muscle in the neck. Thus it has been injured by carrying heavy weights on the shoulder; possibly also by mere muscular exertion, acting directly through the middle scalene muscle. Cases have occurred thus in parturition. It may also be injured by blows upon the chest and by axillary wounds. The nerve is probably also involved in some cases of alcoholic multiple neuritis in which disease paralysis of the external respiratory muscles is not uncommon. Rare cases have been seen to follow typhoid fever and diphtheria.

Symptoms.—Paralysis of the serratus magnus muscle is the only result of injury to this nerve. This paralysis causes an excessive mobility of the scapula, which stands out from the chest wall, giving the patient a peculiar "winged" appearance. This is best shown when the patient elevates the arm: the posterior edge of the scapula then flies out from the chest wall and rotates its inferior angle upward and outward, while the acromion descends. The movement of the arm is thus somewhat

embarrassed, as it is deprived of the basis of support normally given by the shoulder-blade. The chest wall on the affected side is not properly expanded in inspiration. In traumatic cases pain may be felt in the neck. The affection is generally unilateral, but bilateral cases have been reported.

Diagnosis.—The paralysis is easily recognized by the peculiar "winging" of the scapula.

VII. THE CIRCUMFLEX NERVE.

This nerve supplies the deltoid and teres minor muscles, and gives sensation to the skin over the lower and posterior parts of the deltoid. It arises from the posterior trunk of the brachial plexus in common with the musculospiral nerve, and in order to reach its destination it winds around the neck of the humerus—a fact of great clinical significance. It enters the deltoid from the under surface.

Pathology.—The commonest cause of injury to the circumflex nerve is dislocation of the head of the humerus. The nerve may also be injured in fractures of the neck of the humerus. A rare instance is reported by Raymond of injury to both circumflex nerves by pressure during sleep, the patient lying on his back with the arms elevated and the hands clasped behind the neck. One or both nerves may be injured also during surgical anæsthesia. This nerve is not usually the seat of spontaneous or primary neuritis, although it may be affected in lead poisoning and in diabetes. It lies too deep to be often injured by external causes. In cases of arthritis, rheumatic or otherwise, of the shoulder-joint there may be some wasting and paralysis of the deltoid muscle.

Symptoms.—Paralysis of the deltoid muscle is shown by inability to lift the arm from the side and to elevate and hold it above the head. Loss of power in the teres minor causes inability to rotate the head of the humerus outward. As these two muscles take part in various combined movements with the muscles of the upper arm and shoulder, their paralysis is very disabling. The anæsthesia, occupying but a small area, may readily be overlooked, and in fact cannot in some cases be found even by careful search.

Diagnosis.—Paralysis of the deltoid and teres minor muscles is unmistakable from the peculiar disablement of the arm. The greatest risk of error is in cases of dislocation of the shoulder-joint, when the practitioner omits to prepare the patient's mind for the possibility of this complication. A week or more may elapse before the paralysis is recognized, especially if the shoulder has been kept bandaged and pain has prevented movement in the joint. The paralysis is of the flaccid type, and the deltoid muscle may waste rapidly and give the reactions of degeneration. Cases have been recorded in which recovery did not take place for fully a year.

Arthritis causes immobility of the shoulder-joint, but the joint is usually ankylosed and the scapula moves with the humerus. In some of these cases there is more or less atrophy and loss of power in the deltoid muscle.

VIII. THE MUSCULOSPIRAL NERVE.

This nerve is the largest branch of the brachial plexus. It winds around the humerus from within, behind; to the outer side of the arm in a spiral groove beneath the triceps muscle. It divides, near the external condyle, into two terminal branches, the radial and the posterior interosseous nerves. In the upper arm its main trunk supplies branches to the triceps, anconeus, extensor carpi radialis longior, supinator longus, and in part to the brachialis anticus. The interosseous branch, below the elbow, supplies the extensor muscles of the wrist, thumb, and fingers. The musculospiral nerve, and its main branch, the radial, supply sensation to the posterior aspect of the arm, the anterior aspect of the lower part of the arm, and the back part of the forearm, hand, and fingers, except possibly the extreme tips of the thumb and first three fingers, which, according to Richelot, are supplied by the median nerve.

Pathology. — The musculospiral nerve may be injured in various ways. It is exposed to pressure during sleep, when the patient lies with his head upon his arm; this is particularly so in the sleep of alcoholic drunkenness. The nerve is injured occasionally by the pressure of the head of a crutch. Bilateral palsy may be caused in this way. It is also liable to gun-shot and other wounds, but these are rare. The posterior interosseous branch in the forearm is particularly vulnerable to lead poisoning, the well-known wrist-drop resulting. The musculospiral nerve is sometimes injured in fracture of the humerus, in dislocation of the shoulder or elbow, and even by sudden and violent muscular action of the arm.

Symptoms.—These depend upon the seat of the injury. If the main trunk is injured high in the upper arm all the muscles supplied by the nerve are paralyzed, including the triceps. This is the case usually in the crutch palsy. In the case of pressure during sleep the triceps usually escapes, and then the paralysis is only in the supinator longus and the extensors of the wrist and fingers; occasionally, however, the supinator longus escapes. When the paralysis is complete the patient cannot supinate the hand, nor extend the hand at the wrist, nor extend the fingers. The resulting wrist-drop is characteristic. A curious result is seen in diminished power of flexion of the hand and fingers, not that the flexors are truly paralyzed, but according to a physiological law that when the antagonistic muscles are paralyzed the protagonists lose some of their power apparently from failure of a basis of support. This is shown if the hand is passively over-extended, for then the power of the grip is much strengthened. The loss of power of supination excites an effort of compensation in the patient, in which he rotates the humerus outward and presses the arm strongly against the side. The basal phalanges cannot be extended, but the other phalanges, being extended by the interossei, which are supplied by the ulnar, can be extended rather weakly, but best if the basal phalanges be extended passively. As a rule, however, in wrist-drop the fingers are flexed and the thumb turned in and depressed. The paralysis of the supinator longus and brachialis anticus causes some loss of power of flexion of the forearm on the arm, but the latter muscle is not much involved, as it does not receive its whole supply from this nerve. In lead

palsy, as a rule, the supinator longus escapes and may stand out conspicuously among the wasted muscles. The extensor of the metacarpal bone of the thumb may also escape. The muscles most involved in lead paralysis are the extensors of the wrist and fingers.

Sensation may be not much impaired in pressure cases and in lead palsy. When present from a total transverse lesion, anæsthesia is distributed about as follows: When the lesion is high in the upper arm the loss of sensation is located on the posterior part of the arm, the anterior aspect of the lower part of the arm, the back of the forearm, especially on the radial side, and the back of the hand and fingers, except the tips of the thumb and the first three fingers. When the lesion is lower, as in pressure cases, loss of sensation, if present, is only seen below the elbow. As in all peripheral palsies there may be marked atrophy of the muscles, lost tendon reflexes, and reactions of degeneration.

Diagnosis.—The paralysis is so characteristic that a mistake is hardly possible. Lead palsy is bilateral, although occasionally it is worse on one side than the other; it is to be distinguished from the extensor paralysis of alcoholic multiple neuritis by the history; by the pains and the wider extent of the paralysis in alcoholic cases; and by the escape of the supinator longus in lead cases. Pressure cases and traumatic cases generally are unilateral, except in the case of crutch palsy. The history is usually clear.

IX. THE MEDIAN NERVE.

The median is properly called the fellow of the ulnar nerve, as both are flexors of the wrist, hand, and fingers. It arises from the brachial plexus and passes down by the side of the brachial artery. All its branches are given off in the forearm and hand. It supplies all the muscles on the front of the forearm except the flexor carpi ulnaris and the inner half of the flexor profundus digitorum, which are supplied by the ulnar. In the hand it supplies the abductor, the opponens, and the short flexor of the thumb; also the first two lumbricales. Its functions therefore are largely flexor and pronator. It also gives sensation to the radial side of the palm and to the palmar surfaces of the thumb, fore and middle fingers, and the radial side of the ring finger. According to some observers it also supplies the dorsal aspect of the tips of these fingers and the thumb.

Pathology.—The median nerve may be injured in many ways. It is not often involved by pressure during sleep, nor is it injured as often as the ulnar.

Symptoms.—One of the most characteristic symptoms of paralysis of the median nerve is inability to pronate the forearm; this cannot be accomplished beyond the mid-position, and the patient supplements the attempt by rotating the humerus inward. Another characteristic symptom is inability to oppose the thumb to the tips of the fingers; the thumb, in fact, is much hampered in many of its movements, including flexion and abduction. Still other symptoms are impaired flexion of the wrist, which is then done entirely through the ulnar nerve, with consequently marked deviation of the hand toward the ulnar side; and loss of flexion

of the phalanges, except the distal phalanges of the ring and little fingers, which are supplied through the ulnar. The unopposed extensor action of the interossei muscles may cause a subluxation of the joints between the second and third phalanges. Anæsthesia is present on the radial side of the palm, the palmar surfaces of the thumb, fore and middle fingers, the radial side of the ring finger, and the dorsal tips of these fingers and thumb.

Trophic lesions are very common in injuries of the median nerve.

Diagnosis.—This is usually clear from the history of the case and the characteristic distribution of the symptoms. The loss of power of complete pronation, the awkward flexion of the wrist with deviation of the hand to the ulnar side, the disablement of the thumb, and the loss of power of flexion of the fingers, together with the classical anæsthesia, are determinative. The muscular atrophy and reactions of degeneration help to distinguish the case from one of cerebral origin. The trophic lesions, if present, are especially significant.

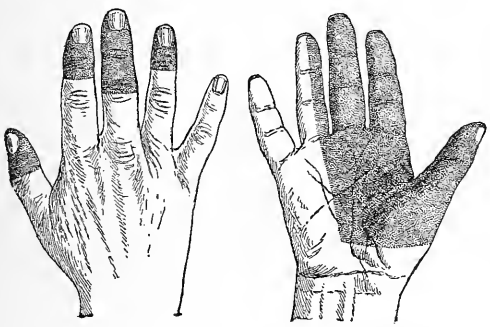


FIG. 400.—Distribution of anæsthesia in paralysis of the median nerve.

X. THE ULNAR NERVE.

The ulnar nerve is one of the two flexor nerves of the wrist, hand, and fingers. It arises from the brachial plexus and passes down the inner side of the upper arm in close proximity to the brachial artery. At the elbow it passes behind the internal condyle, where it can readily be felt in most persons, and where pressure upon it causes a tingling sensation. It supplies the flexor carpi ulnaris, part of the flexor profundus digitorum, the muscles of the little finger, the interosseous muscles, two of the lumbricales, the adductor pollicis, and one head of the flexor brevis pollicis. It gives sensation to the ulnar side of the forearm and hand, the little finger, and the ulnar side of the ring finger. By reason of its distribution this nerve presides over the ulnar flexion of the wrist, the flexion in part of the fingers, the adduction and abduction and other finer movements of the fingers, also extension of the second and third phalanges and flexion of the first phalanges, and the movements of the thumb toward the palm.

Pathology.—This nerve is much exposed to injury, as by wounds, pressure, dislocation, and fractures. It may also be involved in tumors and malignant growths, and in surgical operations. Pressure may occur during sleep, especially during alcoholic sleep and the unconsciousness caused by ether or chloroform; also during parturition.

Symptoms. — Paralysis of the ulnar nerve causes a characteristic disability. Flexion of the wrist is impaired, and on attempts at flexion there is a deviation of the hand toward the radial side. Flexion of the little and ring finger is especially impaired, and the little finger is almost

entirely paralyzed. Adduction of the thumb is lost, hence the patient's inability to grasp objects. Flexion of the first phalanges and extension of the last two phalanges are impaired, but the loss of flexion is less in the first two fingers which are supplied in part by the median nerve. The finer movements of the fingers are abolished. In long-standing cases a characteristic deformity results which is called the "main-en-griffe"; the first phalanges become over-extended, much more so than is possible by voluntary power, and the last two phalanges are strongly flexed. This deformity, however, is not so marked as in cases of anterior poliomyelitis, because the lumbricales of the first two fingers are not involved in ulnar paralysis, being supplied by the median nerve. Muscular atrophy is usually marked; the interosseous spaces are hollowed out, the palm is wasted, and the hypothenar eminence is atrophied. Anæsthesia involves the ulnar side of the arm for some distance above the wrist, also the ulnar side of the hand on both the palmar and dorsal aspects, the whole of the little finger, and the ulnar side of the ring finger.

Diagnosis.—This is comparatively easy, because of the characteristic disablement and deformity. Some care may be necessary in cases due to pressure, as during sleep, or parturition, or surgical anæsthesia. The history of the case, however, is usually clear, and the resulting paralysis is unmistakable. In a case occurring during the delirium of typhoid fever, the paralysis was not detected until the patient's mind became clear and he himself called attention to the loss of power. In irritative injuries, such as gun-shot wounds, trophic lesions may result, such as glossy skin and œdema, and these may be accompanied with burning pain or "causalgia."

XI. THE INTERCOSTAL NERVES.

These nerves are branches of the twelve dorsal nerves. They are separate from each other in the sense that they do not form a plexus. Each nerve runs forward in an intercostal space, and for part of its course it is in close proximity to the pleura.

The intercostal nerves may be variously affected, and they sometimes furnish important indications for diagnosis. This is especially so in diseases of the spinal cord. Thus a spinal meningitis or a spinal tumor may irritate the roots of one or more dorsal nerves, and this may point to the location of the lesion. It may also simulate an intercostal neuralgia. The same is true of spinal caries. In all such organic lesions the diagnosis is to be made by a study of other attendant symptoms. Aneurism of the thoracic aorta may, by irritating a dorsal nerve-root, simulate disease of the spinal cord. Herpes zoster frequently attacks one of the dorsal roots, its point of attack being probably the posterior ganglion, and the pain and characteristic eruption follow closely the distribution of the nerve affected. Intercostal neuralgia is to be distinguished from pleurisy. The affection known as mastodynia, or neuralgia of the breast, is often a puzzling affection, and is commonly seen in neurotic or hysterical women. Fracture of the ribs is usually attended with severe pain in the intercostal nerves, and the diagnosis may require great care, especially where the history of trauma is obscure.

XII. THE LUMBAR PLEXUS.

This plexus is formed from the twelfth dorsal and the first four lumbar nerves. The fifth lumbar nerve does not enter into this formation, but after receiving a branch from the fourth, goes, as the lumbosacral cord, to help form the sacral plexus deep in the true pelvis. This formation of the lumbosacral cord has not a little clinical importance, as will be shown later. The principal nerves arising from the lumbar plexus are the iliohypogastric, the ilio-inguinal, the external and internal inguinal, the anterior crural, and the obturator. The iliohypogastric nerve supplies sensation to the skin of the gluteal and hypogastric regions. It is of little clinical importance. The ilio-inguinal nerve is both motor and sensory. It supplies the internal oblique muscle. It passes out at the external abdominal ring and is distributed to the skin of the scrotum in the male and of the labium in the female, and to the upper inner part of the thigh. The genitocrural nerve is of clinical significance as supplying the cremasteric muscle. It might be involved in psoas abscess, as it penetrates the psoas muscle. The external inguinal or external cutaneous nerve is the sensory nerve of the outer part of the thigh as far down as the knee. Its posterior branch supplies the outer posterior part of the thigh as far as the middle third. The anterior crural and obturator nerves, because of their importance, are described under separate headings. From the nature of their origin and relations, however, they are often involved in lesions of the lumbar plexus.

Pathology.—The lumbar plexus is less frequently the seat of injuries and disease than either the brachial or sacral plexus. Its anatomical position, deep within the body, explains this comparative exemption. The lumbar plexus, being merely a continuation of the nerves which have their origins just within the spinal canal, is closely associated with these parts in its pathology. Falls and crushing injuries may possibly involve the lumbar plexus as well as the intraspinal nerve-roots. Tumors within the body may also cause pressure or irritation. Lumbar and psoas abscess may variously affect these nerve-cords. Finally, parturition may do injury to the anterior crural and the obturator nerves.

Symptoms. — These vary widely according to the particular nerve-cords involved. A good method of diagnosis is carefully to study the motor, sensory, and reflex phenomena *seriatim*, and then by grouping these, to arrive at a conclusion as to what parts of the plexus are involved.

The pain caused by these lesions may be most misleading. Lydston has reported a case of acute lumbar abscess which, by irritating some strands of the lumbar plexus, simulated nephritic colic; the pain was in the iliolumbar region. Psoas abscess may cause pain and paralysis in the anterior crural nerve, which passes through the psoas muscle. It may also involve the genitocrural nerve. Referred or distal pain is sometimes a puzzling symptom from this cause, such as pain on the inner side of the knee and down the inner side of the leg following the course and distribution of the anterior crural and its long saphenous branch. The knee pain due to hip-joint disease is another example, probably due to irritation of the obturator nerve.

The motor and reflex symptoms depend upon the nerves implicated. If the anterior crural is involved the knee-jerk is lost and the extensor muscles of the thigh and leg are paralyzed. The cremasteric reflex may be lost by involvement of the genitocrural nerve. Paralysis of the obturator causes loss of power of adduction in the thigh. Paralysis of the bowel and bladder is not caused by involvement of the lumbar plexus.

Diagnosis.—This has been indicated in the foregoing description of the pathology and symptoms. It is most important to search for gross local lesions, such as abscess, tumor, and vertebral disease. Injuries to the plexus are rare; they sometimes occur as results of child-birth.

XIII. THE ANTERIOR CRURAL NERVE.

This nerve arises from the lumbar plexus and passes out of the pelvis beneath Poupart's ligament on the outer side of the femoral artery. It supplies the muscles on the anterior of the thigh, which act as extensors of the leg; within the pelvis it passes through the psoas muscle, and supplies the iliacus. It gives sensation to the front and inner surfaces of the thigh, and by its main branch, the long saphenous, to the inner side of the leg and foot as far sometimes as the great toe.

Pathology.—The anterior crural may be involved in psoas abscess, as the nerve passes through the fibres of the psoas muscle. Other deep-seated abscesses in the pelvis might also affect it, but lumbar abscess is more likely to affect other and higher branches of the lumbar plexus. Injury to the anterior crural nerve is rare. Fullerton reports the case of a dwarf in which pressure during labor caused transient injury to this nerve; and it may also be hurt in some forms of dislocation of the hip, but only rarely; probably also by a wrench of the psoas muscle.

Symptoms.—There is paralysis of the quadriceps extensor muscle with consequent inability to extend the leg. This paralysis is flaccid, with atrophy, and the knee-jerk is abolished. If the iliacus muscle is involved within the pelvis, there is inability to flex the thigh, which is characteristically shown by the patient lifting the thigh with his hands when asked to cross the lame leg over the other. This movement also is embarrassed by the paralysis of the sartorius. Pain may be present and is experienced from Poupart's ligament to the inner side of the knee, and thence down the inner side of the leg even to the foot and great toe. In psoas abscess this pain is sometimes distal and is relieved by flexing the thigh on the pelvis with the knee kept bent. Anæsthesia involves the anterior and internal surfaces of the thigh and the inner surface of the leg and foot following the course of the long saphenous branch.

Diagnosis.—Psoas abscess may simulate crural neuralgia. The paralysis of the nerve is readily determined by the characteristic loss of power in the iliacus, sartorius, pectineus, and quadriceps extensor muscles, with lost knee-jerk, and by the distribution of the anæsthesia and pain. Irritation of the roots of this nerve might be caused by a tumor in or near the spine, or by disease of the lumbar vertebræ. It may also be paralyzed by aneurism of the femoral artery. Diabetes has been known to cause paralysis and neuralgia in both crural nerves.

XIV. THE OBTURATOR NERVE.

This nerve, after rising from the second, third, and fourth lumbar nerves, and penetrating the inner fibres of the psoas muscle, runs along the inner and lateral wall of the pelvis to the obturator foramen, by which it emerges. It supplies the obturator externus and adductors of the thigh, the articulations of the hip and knee, and the skin of the inner side of the thigh, and, possibly by its communication with the long saphenous nerve, of the leg also.

Pathology. — Because of its deep position injury to the obturator nerve is rare. Some obstetricians believe that it is sometimes involved in puerperal cases, as by the forceps, or the child's head, or in pelvic inflammation. The pain in the knee in the early stages of hip-joint disease is usually ascribed to irritation of the obturator nerve, which sends filaments to both joints; but some orthopædists explain it as due to muscular spasm. Obturator hernia and pelvic tumors may compress the nerve, and it may be irritated by psoas abscess.

Symptoms.—Paralysis of the obturator nerve interferes with crossing the affected leg over its fellow, and with outward rotation of the thigh. The anæsthesia is on the inner side of the thigh and perhaps of the leg, but it may not be marked.

Diagnosis.—This is indicated in the account of the symptoms. The knee pain in hip-joint disease has led to error. In an obscure case it is well to bear in mind the possibility also of a psoas abscess.

XV. THE SACRAL PLEXUS.

The sacral plexus lies deep in the pelvis. It is formed from the lumbo-sacral cord, which arises from the fourth and fifth lumbar segments, and from the three upper sacral nerves and a part of the fourth sacral nerve. It gives origin to four main nerves, the superior gluteal, pudic, small sciatic, and great sciatic.

Pathology.—The sacral plexus may be injured by tumors within the pelvis and diseases of the womb, ovaries, or rectum; by pressure during labor; by wounds; and it may be the seat of neuritis.

Symptoms.—These vary widely, according to the particular nerve-cords involved.

Pain is a common symptom, and may be felt about the buttock, hip, perineum, and even down the thigh and in the leg, foot, and toes, following the course of the great or small sciatic nerves. Paralysis of various muscles is also present, and this paralysis may even present certain types, according to the nerves involved. The bladder and bowel may be paralyzed. For a proper understanding of these cases it is best to describe the various types of the affection.

One of the commonest and most important is the *peroneal* type. This is particularly likely to occur after child-birth. After instrumental delivery, or even after normal labor in rare instances, the patient has pain in the foot and toes; this is soon followed by loss of power in the extensor muscles of these parts, such as the peronei, the tibialis anticus,

and the long extensor of the toes. The patient in walking has foot-drop and lifts the foot high, the toes dragging along the floor. The paralysis is flaccid, the muscles atrophy, and complete reactions of degeneration are seen. The knee-jerks are not abolished. Anæsthesia may be present on the outer part of the leg and on the dorsum of the foot and toes. The explanation of this affection lies in the fact, that the external popliteal or peroneal nerve is a continuation of the lumbosacral cord in the pelvis, and that this lumbosacral cord is particularly exposed to injury by pressure where it runs over the brim of the true pelvis in passing down to the sacral plexus. In these cases there may also be some loss of power in the gluteus medius muscle, which derives its motor supply from a branch of the lumbosacral cord. In most of these puerperal cases the affection is unilateral; occasionally, however, there is a bilateral paralysis, presenting a rather different type.

The form of paralysis varies. All depends upon the particular nerves involved. Occasionally the great sciatic nerve alone is impaired by some intrapelvic lesion, and the case then presents the picture of an ordinary sciatica. A small ovarian tumor has been known to cause pain and loss of power in the buttock. In some of the post-partum cases pain is the chief or only symptom.

Diagnosis. — It is of first importance that the practitioner should determine the cause. When this has once been done the nature of the case usually becomes clear.

The chief difficulty is to distinguish these cases from affections of the cauda equina. The history, however, usually points to an intrapelvic lesion, as in the puerperal and gynæcological cases, and an expert investigation *per vaginam* or *per rectum* will generally detect a local cause if it is present. The examination by the rectum is especially important, as by this means the nerve-trunks can be palpated. Affections of the cauda equina are usually bilateral; while those of the sacral plexus are often unilateral; but this latter rule is not absolute.

Hysteria may simulate these affections, but this psychoneurosis is to be recognized by the mental state, history, and peculiar hysterical stigmata.

Cancer of the rectum, as well as ovarian tumor, has been mistaken for sciatica or some form of pelvic neuralgia. Disease of the sacroiliac synchondrosis may cause obscure and confusing symptoms.

XVI. THE SCIATIC NERVES.

The great sciatic arises from the sacral plexus and passes out of the pelvis by the sacrosciatic foramen, where it is readily accessible to pressure between the great trochanter and the tuberosity of the ischium. It supplies in the thigh the hamstring muscles—the biceps femoris, the semimembranosus, and semitendinosus—and divides just above the popliteal space into two main branches, the internal popliteal and external popliteal (or peroneal) nerves. These two branches supply all the muscles below the knee (both extensor and flexor) and give sensation to most of the leg and foot, the parts not supplied by it being a strip along the inner side of the leg which is supplied by the long saphenous nerve, and the upper part of the calf which is supplied by the small sciatic.

The small sciatic also arises from the sacral plexus and passes out of the pelvis alongside of the great sciatic; it supplies the gluteus maximus muscle (through the inferior genital nerve), and a well-defined area of the skin which includes the buttock, the perineum, and a strip running down the back of the thigh to and including the upper part of the calf.

Pathology. — The great sciatic nerve may be the seat of wounds, inflammation, and tumors. It is sometimes injured in dislocations of the hip, also in forceps deliveries, and by pelvic tumors and even masses of fæces. Inflammation of the nerve constitutes the disease known as "sciatica." Opportunities to examine the nerve are rare in sciatica, but in a few instances on record the nerve-trunk has been found swollen and congested. In the operation for stretching, a similar state has been found. Gout, rheumatism, syphilis, alcoholism, and lead have all been assigned as causes of sciatica. Exposure to cold, especially after great or prolonged muscular effort or a wetting, may cause the attack. Fournier said that it may follow gonorrhœa.

Symptoms.—In organic lesions, such as injury or tumor, the symptoms are characteristic. Pain is an early symptom, sometimes with twitching of the muscles and drawing up of the leg. If the nerve is severed or in any way completely paralyzed, there is loss of power in the hamstring muscles and in all the muscles below the knee, with anæsthesia on the outer side of the leg, dorsum of the foot, the toes, the sole, and a portion of the inner and back part of the leg. The regions that are exempt are a narrow strip on the inner side of the leg, supplied by the long saphenous nerve, and the upper part of the calf, supplied by the small sciatic. The muscles atrophy, and reactions of degeneration are present. The knee-jerk may not be lost. Trophic ulcers sometimes occur.

Pain is the common and sometimes the only symptom in sciatica. This pain is severe and neuralgic in type, especially felt about the upper and back part of the thigh. It may also extend down along the course of the nerve and be particularly intense in the calf of the leg and even in the foot and toes. Any unusual motion in the leg may aggravate it, as suddenly bending the thigh. In such cases the forcible action of the pyriformis muscle probably causes pressure on the hypersensitive nerve-trunk at its exit from the pelvis. In the early stages the pain may be paroxysmal; later it is constant, dull, and aching, or even severe and shooting. Sensitive points are found at the exit from the pelvis, behind the knee, at the head of the fibula, and on the back of the foot. The trunk of the nerve in the thigh is usually extremely sensitive to pressure. Firm pressure between the great trochanter and the tuberosity of the ischium is usually most painful.

Paræsthesia is sometimes observed, as burning and tingling in the areas of distribution of the sensory nerve endings. Anæsthesia is not common, and when present may escape detection if not carefully sought for.

Motor involvement is not common in sciatica. There is usually no true paralysis of muscles, no atrophy, or reactions of degeneration. The knee-jerk, as a rule, is not affected. The Achilles-tendon jerk may be lost. Inhibition of motion is not uncommon, but this is due to pain. The gait is affected for the same reason, and there may be

slight scoliosis. The muscles may become flabby and wasted from disuse. Trophic lesions are rare. Herpes may occur.

The small sciatic nerve may be injured along with the great sciatic at the point where they emerge together from the pelvis. Instances in which it is injured alone must be very rare. Its paralysis causes loss of power in the gluteus maximus muscle, which is shown in inability to rise from a sitting position. This paralysis, however, is due to involvement of the inferior gluteal nerve, which, according to Gray, is closely associated with, rather than a branch of, the small sciatic. Anæsthesia is present on the buttock, the perineum, and the back part of the thigh and popliteal space as far as the upper third of the calf.

Diagnosis.—If there is some organic cause, as injury or tumor, the history of the case and a careful physical examination usually reveal it, and, with the characteristic motor and sensory symptoms, disclose the nature of the case. It is said that flat-foot may cause sciatic pain.

It is easy, as a rule, to recognize sciatica. Pelvic abscesses, disease of the uterus and ovaries, large fecal accumulations, and especially cancer of the rectum have been mistaken for sciatica. Bone disease in the lumbosacral spine may simulate sciatica by irritating the nerve-roots. Hip-joint disease is to be distinguished by the pain on moving the joint, restriction of motion, alteration in the length and position of the limb, tilting of the pelvis, and the other classical symptoms. Obscure cases always call for a rectal examination or vaginal examination. Appendicitis may cause deep-seated pains in the pelvis, but is not likely to be mistaken for sciatica. In sciatica the most distinctive symptom is the pain on pressure on the nerve-trunk. A sign pointed out by Lasègue is of diagnostic value—when the patient lies on his back and the extended limb is elevated, pain is not felt until an angle of about 90° is reached, and this pain is then relieved by bending the leg at the knee. The disease is unilateral in the vast majority of cases, bilateral sciatica being usually symptomatic of organic disease, such as myelitis, tumor, spinal caries, or affections of the cauda equina. Still, cases of genuine bilateral sciatica have been seen, especially in constitutional diseases, as gout and diabetes.

XVII. THE INTERNAL POPLITEAL NERVE.

This nerve is a continuation of the great sciatic. It supplies the flexors of the foot and toes (the calf muscles and muscles of the sole), and gives sensation to the outer part of the back of the leg, to the sole, and in part to the toes.

Pathology.—The internal popliteal, or posterior tibial nerve as it is called in its extension down into the leg, is so deeply situated that it is but seldom injured. It shares, however, in some diseases with other nerves, especially such affections as cause muscular atrophy and club-foot. In many of these cases, however, the seat of the lesion is in the anterior gray matter of the cord rather than in the nerve itself. A case of injury to the nerve from the Matas operation for popliteal aneurism has been observed by the writer.

Symptoms.—Paralysis of the flexor muscles of the foot—the soleus, gastrocnemius, and plantaris—which are supplied by this nerve, causes the form of club-foot known as valgus, sometimes associated with cal-

caneus. Thus flexion of the foot is impaired, and the unopposed peroneus longus pulls the outer edge of the foot upward. The patient tends to walk on the inner edge of his foot and on the heel, and he cannot lift himself on his toes. When the nerve is injured, loss of sensation is found on the lower part of the back of the leg and on the sole and outer aspect of the foot.

Diagnosis.—The diagnosis is easily made from the characteristic distribution of the paralysis and anæsthesia.

XVIII. THE PERONEAL NERVE.

The peroneal or external popliteal nerve is in appearance a branch of the great sciatic arising in or just above the popliteal space, but according to some observers its fibres are really derived from the lumbosacral cord in the pelvis, as is proved by the occasional high division of the great sciatic within the pelvis, in which cases the peroneal is seen to be a continuation of one of these branches, which itself is a continuation of the lumbosacral cord. It passes into the leg behind the head of the fibula, where it may easily be palpated and where it is especially exposed to injury. It supplies the extensor muscles of the foot and toes, and gives sensation to the lower and outer part of the leg, the inner and outer parts of the ankle and foot, the dorsum of the foot, the inner side of the great toe, and the adjoining sides of the other toes.

Pathology.—This nerve is sometimes paralyzed by trauma acting upon it near the head of the fibula. It may also be involved in fracture of the fibula. The peroneal is particularly liable to involvement in cases of alcoholic multiple neuritis in association with other nerves; and in the polyneuritis following typhoid fever this nerve, as well as the ulnar, is apt to be conspicuously affected. There is also a form of muscular atrophy—the so-called Charcot-Marie-Tooth type—in which the muscles supplied by the peroneal nerve suffer especially. The disease is likely to begin in these muscles, but later the hands and forearms become affected. It probably depends upon a peripheral neuritis, and is sometimes a familial affection. Thus Ormerod observed three cases of this disease following measles in one family. The distal muscles are more affected than the proximal. In puerperal palsy the fibres going to form the peroneal nerve may suffer from pressure of the child's head on the lumbosacral cord and sacral plexus. Among rare causes of peroneal palsy are the application of an Esmarch tourniquet, and the pressure of stilts. It is a curious fact that for some obscure reason potato-pickers and others who work in a stooping position sometimes get paralysis of this nerve (Oppenheim).

Symptoms.—There is loss of power of extension of the foot and toes, with consequent foot-drop in walking. The first phalanges of the toes may be flexed by contracture of the interossei. The anæsthesia is on the outer side of the lower leg and ankle, the dorsum of the foot, and the toes. According to some observers the inner side of the ankle is anæsthetic.

Diagnosis.—The diagnosis is easily made from the characteristic distribution of the symptoms. The paralysis of this nerve is distinguished from that of its main trunk, the sciatic, by the escape of the parts supplied by the internal popliteal, and of the hamstring muscles.

GENERAL NERVOUS DISEASES.

1. CHOREA; SYDENHAM'S CHOREA

St. Vitus's Dance.

A disease of children and young adults, characterized by continuous, irregular, involuntary muscular contractions and psychical derangements.

This substantive affection has nothing in common with a number of other diseases unfortunately described as chorea or choreiform, except abnormal muscular movements.

Etiology. — Predisposing Influences. — The disease appears in successive generations in certain families. The readily transmitted neurotic constitution plays a more important rôle. The nervous, excitable children of nervous parents are especially liable to chorea. Chorea is particularly a disease of childhood and adolescence. It is rare before the fifth year and after the fifteenth. The cases in early adult life almost always occur in women. The disease occurs about three times as often in females as in males. Chorea is more common among the children of the poor, but is frequently observed among those living in affluence. It is rare among negroes. The disease is relatively frequent among bright, intelligent school children, especially girls, between ten and fifteen, who are encouraged by their teachers to unreasonable and unnatural application to study. Anæmia and general poor health often precede the attack, but, on the other hand, it frequently occurs in well-nourished children. The disease frequently develops in chlorotic girls. *Scarlet fever* is sometimes followed by chorea. This sequence also occurs as regards whooping-cough and other diseases of childhood; but there is no satisfactory evidence of a causal relation between these infections and chorea. In older persons chorea has been observed after gonorrhœa and sepsis. Chorea frequently develops shortly after an attack of acute rheumatism, and in a group of cases the arthritis is so mild and the choreic symptoms are so prominent as to justify a doubt as to whether or not rheumatic fever has actually been present. In other cases there is a history of rheumatic fever months or years before the development of the chorea. Under either of these conditions the evidences of endocarditis or chronic valvular disease may or may not be present. The endocardial murmurs present in a case of chorea may be due to actual valvular lesions, to irregular action of the heart, or to anæmia. A woman suffering from chorea may become pregnant; and the disease may develop during pregnancy or after parturition. It has been observed in repeated pregnancies. It begins more frequently in the early than in the later months and is often of the severe type—chorea insaniens. About twenty per cent. of the cases in pregnant women terminate in death. The causal relation of pregnancy is shown by the fact that the chorea ceases upon the occurrence of abortion or miscarriage, or delivery at full term.

The Exciting Causes. — In the present state of knowledge it must be assumed that various pathogenic agencies act as the immediate exciting cause of chorea. Among these are: emotional shock, especially

fright; mental shock, particularly in young women; and some conditions associated with rheumatic fever and acute or chronic endocarditis, the nature of which is not understood. The attack has followed a slight injury or a surgical operation and has been attributed to reflex causes, as intestinal worms or genital irritation. It has been stated, but not substantiated, that ocular defects may cause the disease. Finally, the disease frequently develops in the absence of any noticeable cause.

Hypotheses of Chorea.—The view that it is a pure neurosis appears to be widely accepted. The embolic view has some basis of support in the experimental chorea produced in animals by the injection of indifferent substances in fine particles. Cases occur in the absence of endocarditis and without embolism, and with endocarditis but without embolism. That chorea is an infectious disease is an opinion which has the support of many observers. The fact that the attack in an important proportion of the cases directly follows fright or other profound emotional disturbance, and the prominent psychical derangements militate against this view.

Symptoms.—Two forms may be recognized: (1) the ordinary form in which the symptoms are of variable intensity, and (2) the maniacal form—chorea insaniens.

(1) The Ordinary Form.—The onset of the disease is insidious. The child gradually becomes awkward, clumsy, and restless. He cannot sit still. There is a marked change in disposition. He appears to be careless and indifferent, and upon correction has spells of crying or becomes sullen. In the course of some days the characteristic involuntary movements begin. In the mild cases only one hand or the side of the face is affected—hemichorea; in the more severe cases both sides are involved, but one side to a greater degree than the other. In a well-marked case, the child cannot remain quiet, but is in continual motion. The face twitches, the arms are abducted, adducted, rotated, the hands extended, the fingers separated and at once withdrawn and flexed; the head and trunk are rotated and alternately flexed and extended. The gait may be disordered so that progression is unnatural and difficult. These and other movements are rapidly repeated in the most bizarre and disorderly manner, and often with convulsive suddenness. They are manifestly purposeless and constantly vary in extent and direction—"insanity of the muscles." The hands and arms are most affected, the face next, and to a less extent the trunk and lower extremities. The tongue is protruded and withdrawn with a jerking movement; words are uttered with an irregular, jerking cadence, and in grave cases the patient often does not talk at all for hours or even days together. The diaphragm may be involved, a condition which is manifest by irregular, spasmodic breathing. Attempts at voluntary movements increase the involuntary twitchings. There may be transient loss of power in a limb or the entire side. This is usually a mere paresis, but in certain cases it is marked —paralytic chorea.

Emotional influences increase the muscular movements. The consciousness of being under observation almost always intensifies them. As a rule they cease during sleep.

Sensory derangements are not common. In certain cases of hemichorea there is tenderness on pressure and spontaneous pain. Tender points in the line of nerve trunks are rare.

Psychical derangements occur in a majority of the cases. They are usually moderate; sometimes intense. The antecedent neurotic element is to be considered. Irritability, peevishness, and weakness of memory are present. These symptoms often increase, especially in adults, until a condition of mania is established. There is no distinct line of separation between the ordinary forms and those which are most severe.

(2) Chorea Insaniens.—This most intense form does not often occur in children, but is not very rare in young women, and especially in pregnancy. The muscular movements are excessive and continuous. The patient cannot remain standing or lie in bed. There is an uncontrollable jactitation, movement of the eyes and lips, inability to pause to take food or for any purpose, associated with a veritable psychosis with mental confusion, maniacal excitement, and hallucinatory delirium. After a time the patient falls into a state of profound exhaustion, with apathy or melancholia. The temperature may rise to 102°–104° F. (38°–40° C.) and in fatal cases hyperpyrexia has been observed.

The muscles do not waste, their electric excitability is not affected, and the deep reflexes remain normal.

Ocular Phenomena.—The pupils are often dilated, but the light reflex is retained. The muscular twitchings may give rise to transient strabismus. A concentric contraction of the visual field has been observed. The ophthalmoscopic findings are mostly negative, though a few cases of optic neuritis have been reported.

Course of the Attack.—The duration of the disease in children varies usually between six and twelve weeks. There is a remarkable tendency to recurrence, which often takes place in the spring of the year. Two or three attacks are common; as many as five or six have been noted. The tendency to recovery appears to be spontaneous. The chorea of adults runs a longer course. A chronic intermittent form has been described.

Diagnosis.—Direct.—This is, as a rule, made without difficulty. It rests upon the character of the muscular movements, their distribution, the age of the patient, the psychical phenomena, the frequent coincidence of rheumatic arthritis, endocarditis, or chronic valvular disease, and the tendency to spontaneous recovery. The absence of the signs of organic disease of the nervous system is of diagnostic importance.

Differential.—There are several affections occurring in children which present superficial resemblances to Sydenham's chorea.

Multiple Cerebral Sclerosis.—Weakness, incoördination, tremor, and ataxia are characteristic. The gait is spastic paretic; the course chronic, with little tendency to improvement. The reflexes are increased and the intelligence is impaired. Has been described as chorea spastica.

Certain Disorders of Motility Associated with Hemiplegia.—Post-hemiplegic hemichorea is a term used to designate the involuntary movements on one side of the body which manifest themselves in one muscular group after another and give rise to coarse trembling and awkwardness. A prehemiplegic chorea may occur but is much more rare.

Athetosis or hemiathetosis consists in slow involuntary movements chiefly affecting the muscles of the fingers and toes. There are movements of flexion and extension, adduction and abduction, which occur with occasional interruptions both during the waking hours and in sleep, or only upon attempts to use the member, or under excitement. The fingers may be over-extended and spread apart while the hand remains flexed, or some fingers may be flexed and others extended at the same moment.

Hereditary Ataxia; Friedreich's Disease.—The chronic nature of the affection, its occurrence in several members of a family, the ataxia, tremor, scoliosis, and talipes, together with the nystagmus, the scanning speech, and the slow, irregular muscular movements, render the recognition of this disease a comparatively easy matter.

Hysteria.—The movements of so-called hysterical chorea are usually rhythmical and wholly different from those of acute chorea. The stigmata of hysteria are present and the psychical derangements are more marked than in the chorea of childhood. The two affections are sometimes associated.

Prognosis.—In children the outlook is good. Most of the cases terminate in complete recovery. Death may result from exhaustion in the severest cases. The mortality is from 2 to 3 per cent. Chorea in adults, and especially chorea insaniens, is a much more serious affection. In the chorea of pregnancy the death-rate is about 25 per cent. Rapid loss of flesh, delirium, and a rise of temperature are ominous symptoms.

THE CHOREIFORM AFFECTIONS.

Several affections characterized by irregular, involuntary muscular movements are described as chorea. This nosological error is extremely unfortunate and misleading, since these diseases not only have no etiological and but little clinical relationship among themselves, but also none whatever to Sydenham's chorea.

Chorea Major; Epidemic Chorea.—It is a matter of history that at times of religious excitement in the Middle Ages there were extensive popular outbreaks marked by great excitement, gesticulations, and dancing. Under such circumstances pilgrimages were made in the Rhine provinces to the shrine of Saint Vitus at Zebern. This martyr as a saint of succor was invoked for protection against sudden death and against many diseases and distempers, notably chorea, which thus came to be called Saint Vitus's dance. Limited outbreaks of a similar character occurred in the nineteenth century and in this country in Kentucky. The fantastic pilgrimages of the Doukhobors in Manitoba are of this nature. These folk uprisings under the stimulus of religious fervor are hysterical manifestations and have nothing to do with Sydenham's chorea.

Habit Chorea; Habit Spasm; The Tics.—These affections have been misnamed chorea. They have no relationship with Sydenham's chorea.

Momentary grimaces or twitchings of bundles of facial muscles are not uncommon in otherwise healthy adults and are of no clinical significance.

Tic Convulsive; Gilles de la Tourette's Disease.—A psychosis characterized by involuntary violent muscular movements affecting certain muscle groups, as the facial and brachial, or generalized; explosive utter-

ances, which may be inarticulate, sometimes resembling the bark of a dog, or the repetition of words (echolalia), accompanied by involuntary movements, or the repetition of obscene words (coprolalia), or the spasmodic and involuntary imitation of movements (echokinesis), and in many of the cases by curious mental impulses or fixed ideas, as the impulse to touch certain objects (*folie de toucher*), or the obsession of names (onomotomania), or the insane habit of counting with worriment about numbers (arithmomania).

The affection, as a rule, begins in childhood about the time of the second dentition and affects neurotic individuals. The outlook is not favorable, but some of the cases recover. Allied to tic convulsive is the affection known as—

Saltatory Spasm; Static Reflex Spasm of Bamberger; Palmus.—This is probably not an independent disease but in some cases a form

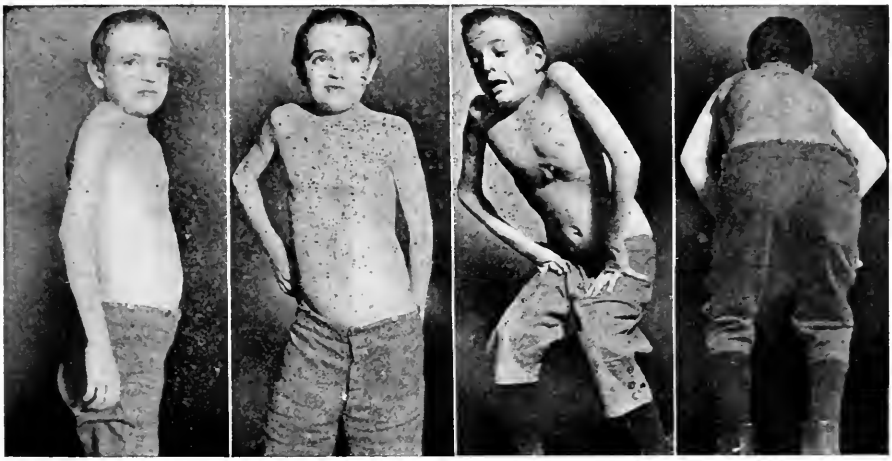


FIG. 401.—Gilles de la Tourette's disease in four phases.—Pennsylvania Hospital.

of tic; in others a manifestation of hysteria; and again a manifestation of increase in the skin- and deep-reflexes. The spasms do not occur when the patient is at rest or in the recumbent posture. When he touches the floor with his feet he begins to hop, jump, and dance about as the result of clonic convulsive contractions of the muscles of the legs and feet, and in particular of the muscles of the calves of the legs. It occurs in both sexes and at any age; in many cases without obvious cause; in others after emotional disturbance or in the convalescence from an infection. It has been observed in dancers as an occupation neurosis. The prognosis is favorable; recovery usually takes place in the course of a few months. The condition occasionally persists for years.

Jumpers.—This form of saltatory spasm has been observed as a local or family neurosis in Maine and parts of Canada. It is characterized by sudden jumping, with outcries, echolalia, and echokinesis. A similar affection has been observed in parts of Siberia—myriachit—and in Java, where it is known as latah.

Chronic Progressive Chorea; Hereditary Chorea; Huntingdon's Disease.—An affection of early middle life, mostly hereditary, characterized by irregular muscular movements, disorders of speech, and irritability and mental weakness gradually leading to dementia. This affection was first described by Huntingdon, of Long Island, who observed it in families in whom it has occurred for four or five generations. The part played by heredity is a conspicuous feature, though cases have been observed in which this etiological factor was absent. Men and women alike suffer. The onset is usually between the thirtieth and fortieth years, and is insidious, without apparent exciting cause. In rare instances it has followed profound and depressing emotion.

The symptoms are motor and psychical. The motor phenomena consist of involuntary, purposeless movements, manifest at first in slight degree in limited muscle-groups, as the hands or face, but gradually increasing in force and extent until all the voluntary muscles are involved. These movements are disorderly and irregular, of wider excursus and less abrupt than in chorea, and cause almost continuous grimaces and gesticulations, increased by excitement and interrupted only during sleep. At first and to some extent throughout the course of the disease they are capable of some degree of temporary control by the force of the will, but presently recur again with renewed violence. In the later stages the gait is much impaired. The body is inclined forward, the trunk sways, and the movement of the legs is irregular, uncertain, and staggering—often arrested for a moment after a few steps. Muscular power is retained until toward the end, when palsies may occur. The deep reflexes are usually increased. Sensation and the special senses are not affected.

Psychical phenomena are irritability, excitability, and depression. Suicide is not uncommon. As the disease advances there are periods of apathy and progressive dementia. Speech is slow, hesitating, and indistinct, the words being slurred and ill-pronounced.

The prognosis is invariably unfavorable. The disease is incurable. Its duration varies from ten to twenty or thirty years. The termination is usually caused by some intercurrent disease or a progressive cachexia.

II. EPILEPSY.

A disease of the nervous system characterized by attacks of unconsciousness, with or without convulsions, recurring at irregular periods. In very rare instances consciousness is not wholly lost.

The following phases occur, independently or in association:

1. *Grand Mal.*—Loss of consciousness with general convulsions, at first tonic, then clonic.

2. *Petit Mal.*—Momentary loss of consciousness without convulsions.

3. *Status Epilepticus.*—Convulsive attacks recur in rapid succession, consciousness not being regained in the intervals between them.

4. *The Psychical Epileptic Equivalent.*—Outbreaks of mania or other mental symptoms, as automatism, take the place of the fit.

5. *Jacksonian Epilepsy.*—The convulsive attack begins in a limited muscle group and may be unilateral and unattended by loss of consciousness; later consciousness may be lost and the convulsions general.

Etiology. — Predisposing Influences. — Age plays an important rôle. In a large proportion of the cases the first attack occurs in early childhood; in the majority before the twentieth year. Many but by no means all of the cases occurring in adult life are symptomatic of a local lesion. In rare instances the disease begins in old persons. Sex is without influence. In children males appear to be slightly more liable; in adults there are more cases among males than females. Direct inheritance is comparatively infrequent, but the children of neurotic families, in which neuralgias, palsies, hysteria, and insanity have occurred, are more liable to become epileptic than the descendants of healthy stock. Paternal or maternal intemperance, especially when associated with syphilis or insanity, is frequently found in the anamnesis. Epileptic convulsions are not very uncommon in the subjects of chronic alcoholism. Epilepsy may occur in syphilitic subjects; more commonly the convulsive seizures are epileptiform and symptomatic of syphilitic disease of the brain. The general convulsions which occur in pathological primary dentition, or in children at the onset of acute infections, in uræmia, in pregnancy, and in chronic lead poisoning cannot in all instances be distinguished from idiopathic or essential epilepsy.

Exciting Causes. — The first attack sometimes follows fright; sometimes an injury; less frequently one of the acute febrile infections. Masturbation has been regarded as a common cause of epilepsy upon insufficient evidence. The fact that certain local irritants give rise to epilepsy, which ceases upon their removal, is incontestably established. Among them are preputial adhesions, collections of smegma behind the corona, intestinal worms, a foreign body in the ear or nose, an irritable scar, phimosis, and a testicle retained in the inguinal canal. It is necessary to assume a strong predisposition in such cases. In some of them the attacks persist after the correction of the offending condition. Overeating and indigestion are very often followed by a fit, and a seizure may follow trauma, gall-stone, or renal colic, or a trifling surgical operation. Epileptiform convulsions occur in heart block—Stokes-Adams disease.

Symptoms.—The recurring fits are the characteristic and in many cases the only feature of the disease. In the intervals the health of the patient is often excellent; in certain cases and in the later stages it may be much impaired.

1. **Grand Mal; Major Epilepsy.** — The attack is frequently preceded by a localized sensory manifestation called an aura. Auræ may be: (a) *Psychical.*—The patient experiences a sensation of strangeness or terror, or a feeling of confusion, or he may fall into a vague, dreamy state, or become extremely gay or furious. (b) *Visceral.*—In this form of aura the sensation is referred to various organs. It is described as the pneumogastric aura. Uneasy sensations in the epigastrium are more common. Sometimes the disagreeable sensation may be intestinal. In other cases it is precordial and attended by anxiety and palpitation. (c) *Peripheral.* —The sensation may begin in the hand or in a finger and extend toward the body before consciousness is lost. (d) *Visual.*—The aura may take the form of phosphenes or color sensations or in rare cases of particular objects. (e) *Auditory.*—Noises or ringing in the ear, curious sounds diffi-

cult to describe, musical tones, or voices. (f) *Olfactory or Gustatory.*—These are rare. They consist of strong odors, almost always unpleasant or disagreeable, or foul tastes and the like. The aura is usually of short duration, the attack coming on in a few seconds. In other cases it may be prolonged.

Premonitory Forced Movements.—In some cases the aura does not occur, but in its place there are definite forced movements. The patient turns rapidly or twirls upon his toes, or runs a few steps or even to and fro a number of times.

The Epileptic Cry.—At the onset of the attack the patient very often utters a loud scream or yell.

The attack is instantaneous. Consciousness is at once completely lost and the patient falls as if shot. Slight injuries are common, and grave, even fatal accidents, as fracture of the skull, sometimes occur. The FIT consists of three stages,—(1) tonic spasm, (2) clonic convulsions, and (3) coma.

(1) *Tonic Spasm.*—There is a spastic rigidity of the muscles, including the respiratory muscles, so that respiration is at once arrested. The face is at first pale, then red, and directly bloated and cyanotic; the eyelids are closed or open, the eyes fixed, the pupils dilated, and the iris is irresponsive to light. At the very first there may be a momentary contraction of the pupil, but this soon gives place to dilatation. The head is forcibly extended or turned to one side, the arms are rigidly extended or flexed, the thumbs adducted, and the fingers clinched. The legs may be rigidly extended, or flexed. The tongue may be protruded and caught between the fixed jaws. The fæces and urine may be discharged. This period lasts from a few seconds to half a minute. Toward its close there is tremor, which ushers in the second stage.

(2) *Clonic Convulsions.*—The muscular contractions now intermit. Tremor gives way to rapid and violent spasms. The limbs are tossed about with force. The muscles of the face are violently contracted; the eyes roll from side to side or are turned up, and the eyelids open and close forcibly. The jaw muscles are in violent clonic spasm and the tongue is lacerated by the teeth. A frothy saliva, often stained with blood, is discharged with the violent respiratory movements. Further discharges of urine and fæces may now occur and there is occasionally an ejaculation of semen. This period lasts from one to three or four minutes. The convulsions become less violent and gradually subside and the patient passes into the third stage of the attack.

(3) *Coma.*—The limbs are relaxed and the unconsciousness is profound. The breathing is stertorous and the face flushed but no longer cyanosed. After a time the patient may be aroused, but is dazed and confused and relapses into a deep sleep which lasts for hours.

The following clinical phenomena occur, but are not constant:

Vomiting after the attack. Slight rise of temperature,—one to two degrees of Fahrenheit's scale. In the status epilepticus higher temperatures are observed. Abolition of the reflexes. The conjunctival, corneal, and pupillary reflexes are all abolished during the attack. The deep reflexes may, however, be increased and ankle clonus evoked. Subcutaneous and

subconjunctival extravasations of blood; the former chiefly about the face and neck. Albuminuria during the attack; polyuria subsequently; occasionally an increased urea output.

The aura is not always followed by the fit. When it begins in an extremity, particularly in the hand, the attack may be prevented in some cases by immediate compression of the member by a string or ligature or by energetic pulling or rubbing of the part.

2. **Petit Mal; Minor Epilepsy.** — Momentary loss of consciousness is the chief, often the only symptom. The unconsciousness usually is so brief—a few seconds to half a minute—that the patient does not fall, but directly resumes his occupation or conversation as if nothing had occurred to interrupt it. The face usually becomes pale, the eyes are set and staring, and anything in the hands may be dropped. The tongue is not bitten and involuntary discharges do not occur. In many of the cases slight spasmodic movements of the facial muscles may be noticed.

The attack may take the form of transient vertigo, the true nature of which, in the absence of unconsciousness and twitchings of the lips, tongue, or eyelids, cannot be recognized. Following the attack of petit mal there may be slight incoherency or automatic actions, such as beginning to undress, spitting, or rubbing the face or head. The patient may in other cases fall without the occurrence of convulsions. There may be jerking of the limbs, tremor, or sudden visual sensations. The significance of these phenomena is revealed by their occurrence in persons who manifest well-characterized attacks. An aura is rare and many patients are unaware of the occurrence of the attacks. As a rule convulsions gradually develop, and in many of the cases petit mal and grand mal are associated.

3. **Status Epilepticus.**— The patient passes from one convulsive seizure into another, consciousness not being regained in the intervals. The pulse and respiration are rapid, the temperature rises, and the attack frequently terminates in death. Hyperpyrexia is not uncommon and temperatures of 107°–111° F. (41.7°–43.9° C.) have been observed. Status epilepticus may last two or three days.

4. **Psychical Epileptic Equivalents.** — Remarkable psychical disturbances sometimes take the place of the fits or alternate with them. The patients perform extraordinary and apparently premeditated acts of which they have no knowledge or subsequent recollection. They run about, wander away, throw away their clothing, commit violent, even murderous assaults without motive and without self-restraint. These psychical states are not easily distinguished from the maniacal states which sometimes follow the attacks — postepileptic delirium. These conditions may last for hours or for several days. They usually come on suddenly without premonitory symptoms.

Other derangements which are regarded as equivalents are sudden and profuse sweating, sudden sleep (narcolepsy), the automatic repetition of meaningless words or phrases (verbigeration), and attacks of general tremor with impaired consciousness.

5. **Jacksonian Epilepsy; Cortical, Partial, or Symptomatic Epilepsy.** —Consciousness is not at first lost. It may be preserved throughout the attack. The attacks are the result of irritative lesions of the motor

zone, as tumor, inflammatory softening, acute and chronic meningitis, hemorrhage, abscess, and trauma. They occur also in general paresis. The spasm begins in a limited group of muscles of the face, arm, or leg. Numbness or tingling is followed by limited spasm, which extends and involves a limb or the side of the face. The spasms may be localized for a long time; but ultimately tend to become general. Posthemiplegic epilepsy is of the Jacksonian type. The convulsions may for a long time be confined to the paralyzed side, beginning in the hand or foot without unconsciousness.

The mental state of the epileptic is often normal. More commonly there is absence of self-control, associated with depression and irritability. As the disease progresses there is often impairment of intelligence and memory. The seizures may occur daily for a period and then at longer intervals; in other cases they may occur only once in many months. It is common for them to recur at irregular intervals of two or three weeks. The attacks of petit mal may occur many times a day. The attacks are more common by day than at night. Nocturnal epilepsy may go on for a long time without being recognized. In women the attacks frequently occur at or near the menstrual period.

Diagnosis.—DIRECT.—Major epilepsy declares itself by the aura, the cry, the instant loss of consciousness, and the consecutive tonic and clonic spasm followed by coma or stupor. The relaxation of the sphincters and the bitten tongue are distinctive. The recurrence of the fits at irregular periods is an essential feature.

The minor attacks are characterized by momentary loss of consciousness or vertigo. Twitching of the facial muscles is suggestive. Their frequent recurrence and association with grand mal are diagnostic. Status epilepticus occurs as a culminating condition to be known not only by the recurring convulsions and intervening coma, but also by the history of the case. Psychical equivalents of the attack can only be recognized in the light of the anamnesis. There is nothing distinctive in the delirium, mania, or delusions. The diagnosis of Jacksonian epilepsy rests upon its characteristic symptoms, local spasm, and retention of consciousness. The recognition of the peculiar condition of which it is symptomatic is often difficult.

DIFFERENTIAL.—The importance of the distinction between true epilepsy—the so-called idiopathic form—and symptomatic or epileptiform convulsions cannot be over-estimated. To the latter belong convulsions of the Jacksonian type. The diagnosis of epilepsy can never be made from a single attack, particularly when it has not been seen by the physician. *Syncope* may be mistaken by the untrained for epilepsy. The unconsciousness is not so complete as in epilepsy, nor is it preceded by an aura, accompanied by tonic and clonic convulsions, involuntary discharges, or followed by coma or automatic actions. *Ménière's disease* may simulate an epileptic seizure, but aural phenomena are present and the characteristic symptom-complex of epilepsy is absent. *Toxic Conditions.*—The general convulsions of pathological puerperal states, uræmia, lead intoxication, and other toxic conditions are not to be confounded with epilepsy. The underlying causes of these symptomatic seizures are usually plainly manifest. When they occur in individuals previously epileptic, their essential nature may remain in doubt.

General Convulsions of Infancy; Eclampsia.—These are due to direct irritation of the cerebral cortex, reflex irritation, and toxic influences. In early infancy epilepsy is the least obvious diagnosis and should never be made until other causes have been excluded. In older children the seizures may be symptomatic of peripheral irritation or intestinal worms, or they may replace the chill which in adults marks the onset of an acute infectious disease. When they are repeated, only prolonged observation will justify a positive diagnosis. Complete restoration to the usual health in the course of a few hours, especially in a child who presents the stigmata of degeneration, is an important point in favor of a diagnosis of epilepsy.

Coarse Cerebral Lesions.—Cerebral focal disease and meningocortical lesions may cause symptomatic or partial epilepsy. When the convulsions rapidly become general they may closely simulate general epilepsy. In truth the border line between the two forms is not always sharply drawn. The symptomatic convulsive seizures that occur in general paresis are commonly unilateral and not attended by loss of consciousness.

Hysteria.—(See p. 814.)

Hystero-epilepsy.—The rare forms of major hysteria are attended with recurrent convulsions, which may be readily distinguished from repeated epileptic seizures or the status epilepticus by the emotional prodromes, the hysterogenetic points, globus, contortions, histrionic poses, and hallucinations. There are mixed forms of hysteria and epilepsy and transitional forms. In other words the hysterical person may be also an epileptic.

Simulated Epilepsy.—The normal light reflex, absence of dilatation of the pupil, the absence of instantaneous pallor at the onset of the attack, the lack of the characteristic cyanosis and flushing, and the condition of the patient after the attack are of diagnostic value. The convulsion may be feigned, but the tongue is not bitten and postepileptic coma and mental confusion cannot be imitated.

Prognosis.—In the great majority of cases epilepsy is an incurable disease. No case can be looked upon as having recovered unless there has been complete freedom from the attack for a period of several years. The outlook is less favorable when the disease begins in infancy or childhood than in the cases in which it begins at puberty. When it begins between the twentieth and thirty-fifth years complete recovery is rare. The more frequent the attacks and the longer the period in which they have continued to recur, the more unfavorable the prospect of recovery. The prognosis is unfavorable in degenerates and those suffering from inherited or acquired mental disease. Recovery is more rare in females than in males. The severity of the individual attack has no direct relation to the prognosis, except that the outlook is less favorable when the disease begins as petit mal, and that in the status epilepticus about one-half the cases die in the attack. Epileptics are frequently short lived. The attack in itself is not especially dangerous to life. In very rare instances asphyxia or cardiac rupture may occur. The seizure is attended with the risk of serious, even fatal injury which may result from sudden loss of consciousness. The patient may fall from a height or under a vehicle or into a fire or water. The outlook is more favorable in symptomatic convulsions than in essential epilepsy.

III. HYSTERIA.

Hysteria is a psychoneurosis, in which the mental state induces and dominates a great variety of physical symptoms. Its name, which is derived from the Greek word for the uterus, indicates an error which has prevailed for more than two thousand years, for hysteria has no necessary relation with the womb. It occurs in men and in young children as well as in women. Heredity is a common cause, as was demonstrated by Briquet. Next in importance is trauma, and then mental excitement and moral shock. Toxæmia, metallic poisoning, and acute disease all act as occasional causes.

Pathology.—Hysteria has no recognized pathology. It is a so-called functional disease. The theory that it depends on minute structural changes in the neurons is possibly correct, for all function depends upon structure, but we have no way of ascertaining these changes.

Symptoms.—Hysteria is most common in children and young adults. It rarely appears after middle life.

The French divide the symptoms into two great classes,—the paroxysmal and the interparoxysmal.

The *paroxysm*, or fit, is divided into four periods. Prodromes or auræ may usher in the first period; the former usually are changes in temper and disposition, the latter are the *clavus*, a circumscribed pain in the head, the *globus hystericus*, a sense of a ball rising in the throat, and ovarian hyperæsthesia. Other and more rare prodromes and auræ are seen.

The *first*, or epileptoid, period is marked by a sudden, tonic spasm, in which the patient lies rigid or even in opisthotonus, with hands clenched, eyes fixed or even crossed, and arms extended in the position of a cross; the breath is labored, the pulse slightly accelerated, and consciousness is obtunded, but seldom or never entirely lost. The tongue is not bitten, the pupils are not affected, incontinence of urine does not occur, and the patient does not injure herself in falling. It is this stage which most closely resembles epilepsy. But the clonic spasms which supervene are not exactly like those of epilepsy; they are usually more irregular, and often have something of a voluntary aspect. The eyelids present a slight tremor.

The *second* is called by the school of Charcot the period of "clownism." The patient throws herself into grotesque attitudes; she seems as one possessed, and indeed she was believed, in the Middle Ages, to be controlled by a demon. Extreme opisthotonus is one of the commonest positions assumed by the grand hysteric.

In the *third* period the patient seems to act a part; she is dramatic, sometimes pathetic, always extreme. This is the histrionic stage, in which the conduct is evidently the mirror of certain mental states. It has been greatly elaborated by the French school, and by means of hypnotism and suggestion has been not a little overdone.

The *fourth* period is that of delirium—so-called. The patient subsides into a state of weeping and declamation; sometimes there are spells of laughing.

The hysterical fit is not always typical; there are aberrant or abortive forms. In this country we seldom see the whole tableau. The first

period is the commonest, with a brief histrionic display, followed by a crisis of weeping and laughter. Among the very rare aberrant forms are ecstasy, somnambulism, catalepsy, and trance. Tourette and Cathelineau have tried to show that the nutrition is affected in a characteristic way; there is loss of weight, with increased excretion of urea, but during the lethargic trance-like stage the urea diminishes. Some of the sensational stories of the dead returning to life, as in the case of Lady Russell, who revived at her own funeral, were doubtless founded on cases of hysterical trance.

The *interparoxysmal* symptoms are motor, sensory, and visceral.

The *motor* symptoms are paralysis, contracture, tremor, and incoördination.

Fig. 402.—Hysterical hemiplegia, showing glossolabiobrachial spasm of left side.—Stewart.

The paralysis may take the form of a monoplegia, a hemiplegia, a paraplegia, or a total palsy. It is sometimes limited to one or a few muscles, as of the hand, arm, face, tongue, pharynx, or larynx. The paralysis in the extremities is likely to be accompanied with contracture; the deep reflexes are not abolished, nor is there true muscular atrophy, or reactions of degeneration. The paralysis is not, as a rule, limited to the distribution of particular nerve-trunks; in other words, it is *central*, not peripheral. The paralyzed part may become œdematous, blue, and mottled, especially in traumatic cases. Hysterical paralysis is usually persistent for long periods, but occasionally it is transitory and recurring; and mild grades may even be transferred from side to side. Sometimes a permanent cure is effected suddenly. In hemiplegia the leg is usually more paralyzed than the arm, and the face and tongue are not affected. In paraplegia there may be anuria but not incontinence. In total paralysis all four limbs are involved, but the face and trunk escape. Chevalier was able to find only 21 authentic cases on record.

Contracture sometimes coexists with paralysis, but the rule is not universal. The paralyzed limb is not always contractured, neither is the contractured limb always paralyzed. The contracture does not always relax during sleep, but it relaxes under ether or chloroform. It may come and go; in some cases it is painful, and it can sometimes be re-established by pressure on the main nerve-trunk. Surprising cases are on record of long-persisting hysterical contractures. They sometimes

follow trauma, or the grand convulsion, or sudden shock, and are often accompanied with other stigmata, such as aphonia, anæsthesia, etc.

Tremor is of several types: the most common is that which resembles the intention tremor of multiple sclerosis; of wide amplitude, absent during repose, increased by volition, and likely to be caused by trauma, or by metallic poisoning (lead and mercury). The "type Rendu" closely resembles this tremor, except that it may persist during repose, and is merely aggravated by volition. Dutil has also described a very fine tremor of from 8 to 12 vibrations to the second. Westphal's pseudosclerosis is doubtless a form of hysterical tremor.

Astasia-abasia is one of the curios of hysteria. It consists of a loss of power of standing (astasia) and of walking (abasia). There is no true loss of power or any necessary loss of sensation, and when the patient sits or reclines there is usually no incoördination. Progression on all fours is even possible. The gait consists in a series of wild, incoördinate movements of the legs, with alternate bendings backward and forward of the body. But little progress is made, and the patient requires support on each side. There is sometimes an alternate stiffening and relaxation of the back and legs, causing a tendency to opisthotonus and a rising on the toes. Astasia-abasia is most likely to be caused by trauma and emotion, and is most frequent in young persons.

Fig. 403. — Hysterical contracture. — Lloyd.

Sensory changes consist of anæsthesia, hyperæsthesia, and paræsthesia. Anæsthesia is of various kinds, such as hemianæsthesia, segmental anæsthesia of a limb, and anæsthesia in patches. Hemianæsthesia is usually complete; that is, it extends from the crown of the head to the sole of the foot, and is often accompanied with anæsthesia of the mucous membranes of the eye, nose, tongue, mouth, and throat. It is sharply delimited at the median line, and can sometimes be transferred from one side to the other by suggestion. The special senses, sight, hearing, smell, and taste, may be involved on the affected side. Segmental anæsthesia of a limb is not uncommon; the area is sharply delimited above by a transverse boundary line, thus presenting the shape of a stocking or a gauntlet. Irregular anæsthesia in spots and curious geometrical figures, scattered at random over the surface of the body, and changing repeatedly, is quite characteristic. These various sensory stigmata play, and have played, an important rôle in hysteria. In the Middle Ages, during the witchcraft crazes, they were known as the "marks of the devil" (*stigmata diaboli*). The anæsthesia of hysteria, in whatever form, is very real and very profound, and even involves the

subcutaneous tissues and the nerve-trunks. The patient may not know of its existence, and it requires careful tests for its demonstration. According to Pitres hysterical anæsthesia is never isolated tactile anæsthesia; in other words, one or more of the other forms of anæsthesia—such as analgesia, thermo-anæsthesia, and even electro-anæsthesia—are always present, and in some cases there is a loss of sensibility to all modes of sensation. Loss of muscular sense is a rare phenomenon. The electro-æsthesia may be preserved, however, when all other modes are lost, but not inevitably. Hyperæsthesia is found in certain zones or territories, as along the spine, and especially in the ovarian region, where pressure may excite a fit or cause other hysterical stigmata. These are the so-called hysterogenous zones. Paræsthesia, consisting of altered sensation, is not so common or significant.

Of the special senses the eyes present the most important changes; there is contraction of the visual fields, and reversal of the color fields, the red being larger in extent than the blue. Hemianopsia is rare. Total blindness or amaurosis has been noted. Blepharospasm, or spasm of the orbicular muscle, is seen, and may be mistaken for paralysis (ptosis), but true paralysis of any of the ocular muscles is extremely rare. Lloyd has seen true iridoplegia in hysteria. Spasm of one or other ocular muscle may cause strabismus, and be mistaken for paralysis of the opposing muscle.

Hysterical deafness, anosmia, and loss of taste are occasionally seen.

Visceral symptoms of various kinds are observed. Anuria is not uncommon; the patient may even require to be catheterized—a bad procedure in hysteria, since it tends to confirm the weakness. Incontinence is not seen. Hysterical vomiting—*anorexia nervosa*—consists of a regurgitation of food, rather than a true vomiting. The food is rejected before it reaches the stomach in most cases. The curious habit known as *merycism*, or chewing the cud, in which the patient regurgitates and remasticates the food, is a closely allied symptom. In hysterical vomiting there is no nausea, but the patient may emaciate and present other hysterical stigmata; in some cases, however, the nutrition is wonderfully preserved. Rapid respiration is sometimes seen, the respirations running as high as seventy or more to the minute; but the pulse is not accelerated, the color remains good, and there is no real dyspnœa. The breathing is shallow or panting. Occasionally, without increased breathing, there is tachycardia, which may even persist in spite of prolonged rest in bed. Persistent cough is sometimes a perplexing and exasperating symptom, as are also bouts of yawning. The cough is unattended with the physical signs of lung disease; and the yawning occurs in paroxysms, much exaggerated and prolonged, but not noisy. Aphonia is not rare. The patient may talk in a whisper, but sometimes is quite speechless, and even voiceless. Instances are reported of the natural voice returning during laughter or even during sleep, but a cure does not necessarily follow. Phantom tumor can be caused by contracture of the abdominal muscles. The French writers describe pyrexia, or pseudopyrexia. Some of the temperatures recorded are quite incredible. The subject requires further study.

The *psychical* state in hysteria is most important, for it is the essential one. To investigate it requires expert knowledge and skill, and it is sufficient here to say that one of its chief features is suggestibility, in which the patient's mind is peculiarly impressionable to outside influences. Freud's theory of "repressed emotions" need not be discussed here.

Diagnosis.—To begin with, the practitioner should disabuse his mind of the vulgar prejudice that hysteria is a simulated disease. The hysterical patient is not a humbug or malingerer. The affection is very real, and these patients are genuine sufferers. Many of them are useful members of society; some of them, to be sure, are weaklings, and a few are even degenerates, but they are none the less entitled to consideration.

It is commonly said that hysteria simulates all diseases; but the truth is that it simulates none exactly. There is always something *sui generis* in the hysterical stigmata. On the other hand, hysteria itself is sometimes simulated by designing persons, especially young women, but the counterfeit is usually detected with ease. No person can simulate successfully, especially for long periods, the paralysis, the contracture, the anæsthesia, or in fact any of the more important stigmata of hysteria. If any one doubts this, let him try to simulate contracture of the arm for a week.

There is often confusion between hysteria and neurasthenia, especially in the traumatic cases; and, in fact, many of the so-called traumatic neuroses are hysterical. The two conditions merge into each other, and the dividing line is not easily determined in some cases. The mental state of suggestibility is highly characteristic of hysteria; also the tendency of symptoms to come and go, and to be influenced by hypnotism. In genuine neurasthenia this is not so marked. Moreover, in neurasthenia we do not see the characteristic permanent stigmata of hysteria, such as the paralyses, anæsthesias, aphonia, anuria, etc.

The hysterical fit can closely simulate that of epilepsy, but there is no biting of the tongue, no frothing at the mouth, no injury to the person, no involuntary passage of urine, and the pupils are not affected. The state of the pupils may be a criterion of great value, for in the epileptic fit the pupils dilate after a momentary contraction in the tonic stage, and do not react to light. In mild cases, however, such as petit mal, the pupil in epilepsy may respond to light. The state of the consciousness is appealed to by many as a true test of epilepsy, but it is not always reliable. Doubtless the profound unconsciousness of grand mal is not seen in hysteria, but in petit mal the consciousness is often but momentarily confused, hardly lost, and these are cases that might be simulated by hysteria. Yet hysteria is not usually so momentary as petit mal; and in the hysterical fit, it is true that, as a rule, consciousness is not so completely abolished as in grand mal. The hysterical fit can sometimes be induced by pressure on the ovarian region, and it is likely to be followed by hysterical stigmata, such as anæsthesia or even paralysis. Finally the last three periods of the hysterical fit are determinative, for they are never seen in epilepsy; but neither are they always seen in hysteria. It is important to bear in mind that hysteria and epilepsy can coexist in the same patient and present a confusing picture, but the crises are separate.

The diagnosis of the various permanent stigmata, such as paralysis, tremor, astasia-abasia, anæsthesia, vomiting, phantom tumor, etc., has been indicated in the description of those symptoms. Taken alone, they sometimes closely simulate organic disease, but they are usually associated with other hysterical stigmata, and this fact and the history are determinative. Ovarian pain, hysterical in origin, is not seldom mistaken for evidence of organic disease, and women are thus subjected to operation and needless mutilation. To guard against this too common error the practitioner should study his case well with reference to other hysterical stigmata.

IV. HYPNOTISM.

It is impossible to define hypnotism, or hypnosis, in satisfactory terms. It is a mental state, resembling, but not identical with, sleep, in which consciousness is variously affected, but in which the mind usually remains open to suggestion, especially from the person who stands in relation of hypnotizer to the patient. In the opinion of some good observers hypnotism is merely a form of induced hysteria.

Pathology. — Like all the psychoses, hypnotism cannot be said to have a recognizable pathology. It is common in hysterical patients, but neurotic and imaginative persons are also susceptible. It is rarely seen in the insane.

Symptoms. — We may recognize here the three stages of Charcot, merely premising that they are not sharply defined in all cases, especially the minor cases.

In the *cataleptic* stage the patient assumes a statuesque attitude, with partially opened eyes, blunted sensibility, and a readiness to receive and act upon suggestion. In many cases it is not possible to proceed beyond this stage, which may be regarded as a minor form of hypnotism.

In the *lethargic* stage the patient passes into a more sleep-like state. The special senses and general sensibility are much impaired, the muscular system is relaxed, and the consciousness is deeply affected. Such patients are not as open to suggestion as in the preceding stage. They are usually highly neurotic individuals, and in some cases they may even pass into a trance.

In the *somnambulic* stage we see a state which has often been miscalled "double personality." The patient's special senses are acute, but she is oblivious of much that transpires about her, although open to suggestion from the hypnotizer. The patient seems to be acting a dream.

Hypnotism can usually be excited by fixing the patient's attention on some particular object and keeping the eyes in a strained and fixed position. The French use bright objects, revolving mirrors, etc. The patient gradually becomes drilled and goes into the hypnotic state on the slightest provocation.

Much speculation, and much that is merely fanciful, has been written about hypnotism, but it is sufficient to bear in mind that it is a psychosis, or mental affection, in which the patient exists in a sort of dream-like state, in which suggestion from without can be made to play a prominent part, and in which hysteria is always an important factor.

V. NEURASTHENIA.

Neurasthenia is defined by Savill as a state of irritable weakness of the entire nervous system, characterized by hypersensitiveness, headache, inaptitude for mental work, disturbed sleep, irritability of temper, restlessness, nervousness, vague pains, and affections of the vasomotor and sympathetic systems. The disease has been much exploited in recent years, for it is not uncommon, especially among the overworked populations of our large cities.

Pathology.—There is no recognized pathology. The nerve centres are at fault, and the most plausible explanation is that these centres are not properly nourished. There is, however, a large mental element in these cases, and, as in all psychoses and neuroses, the affection is usually defined as functional. Among the causes, heredity, overwork, trauma, and the excessive use of alcohol and tobacco are the commonest. Of late years, following Bouchard, importance has been attached to the possible rôle of autointoxication in the various psychoneuroses. The theory is that toxic substances are formed by disordered action in the gastro-intestinal tract, and being absorbed into the blood cause various nervous derangements. The presence of indican, or indol, in the urine is supposed to be a sign of this state, but Buckley cautions against looking upon indicanuria as necessarily equivalent to autointoxication. We do not stop here to discuss the occult theories of Freud and the psycho-analyzers.

From another viewpoint there is a tendency to attach importance to disorders of internal secretions as causes of various so-called functional nervous diseases, and the Abderhalden tests are used by some in an attempt to point out these obscure relationships. But the exact value of these tests is still a matter of debate.

Symptoms.—The symptoms are so multiform that only a generalized view will be attempted here for purposes especially of diagnosis.

The cardinal symptoms are mental ones—the despondency, the inability to apply the mind and to work, and the general nervousness. Some patients are not a little hypochondriacal, and in others an hysterical element is present, but the disease is not essentially either hypochondria or hysteria. Introspection and discouragement are prominent. Upon this psychical state are ingrafted some characteristic bodily ailments.

Headache is not uncommon, and a hypersensitive spine is frequent, especially in traumatic cases and in women. There are sensitive points along the spine, and the least exertion aggravates these and causes a sense of exhaustion. Pain may also be transmitted to the limbs, and sometimes there are bodily pains suggestive of visceral disease. Disturbance of sleep is common; there is either insomnia or broken and restless sleep, so that the patient arises in the morning unrefreshed. Ugly dreams are often a feature of the traumatic cases, the patient seeing again the frightful accident through which he has passed. Nutrition is sometimes greatly impaired, the patients emaciating and becoming anæmic; and they are often bedridden, especially if they are women, and present the appearance of extreme illness. Inability to take and digest a sufficient quantity of food is a

troublesome feature; and there may even be dilatation of the stomach or gastroptosis. On the other hand, some neurasthenics are remarkably well nourished; these are the fat neurasthenics, who are not the least troublesome patients. Morbid blushing and flushing are sometimes seen; and the heart may be accelerated in spite of prolonged rest. Palpitation annoys these patients on slight exertion. Paralysis is not commonly seen, but in the traumatic cases there may be inhibited or impaired movement on account of pain. Genuine paralysis points either to hysteria or to organic injury. The same is true of anæsthesia; it is rare, and its presence is usually due to an hysterical element. Tremor is occasionally seen, and requires to be carefully distinguished from other tremors, such as those of alcoholism and hysteria. The knee-jerks are usually free, sometimes exaggerated, never lost.

Of the special senses the eyes suffer most; there may be eye-strain and pain on using the eyes. Tinnitus, vertigo, and noises in the head, or a sense of fulness, are present in some cases. Some patients have a strange sense of mental vacuity.

Sexual weakness is not unusual in men, and the sexual act causes profound exhaustion, weakness in the back, and headache, which may endure for a day or so.

In this connection it is well to consider briefly the *traumatic neuroses*. They are in fact largely neurasthenic, although a few are purely hysterical. It is too much to say, however, that all these traumatic cases are purely functional. Some of these patients suffer from organic lesions, such as bruises, sprains, and wrenches of the muscular and tendinous attachments, especially of the spine. The so-called "railway spine" is not always purely neurasthenic, but may be in part the result of shock and sprain. The same may be said of some of the obscure injuries to the hip and other joints; there is a large neurasthenic element, but it is very often not the whole story. These cases are of exceptional importance because they lead so often to litigation; and the controversy over them is frequently acute.

All neurasthenics are quickly and easily fatigued, both by mental and physical exertion. Inability to concentrate the mind is not uncommon; and of other mental phenomena the most important are the so-called *obsessions*. They consist of imperative and inhibitive ideas, and are seen in the state known as *psychasthenia*, which belongs rather to psychiatry than to clinical medicine.

Diagnosis.—Since Beard invented the term, neurasthenia has been used very loosely to cover a wide variety of symptoms. When properly guarded, however, the term has a legitimate use, and, although it is hardly capable of exact definition, it covers a symptom-complex which is fairly recognizable; and this has been described above. The diagnosis must depend upon a careful consideration of those symptoms, and the practitioner must not forget that a neurasthenic state often accompanies other diseases, even grave organic ones, or injuries. The only rule is to exercise care and judgment, and to go by a process of exclusion.

The most common error is to confuse neurasthenia and hysteria; in fact, some writers in describing the traumatic neuroses do not hesitate

to include hysterical symptoms indiscriminately with those of neurasthenia. This is wrong. Hysteria is a much more clearly defined disease than is neurasthenia, and it should be kept apart whenever possible. The mental state, and the various stigmata, such as paralysis, anæsthesia, contracted visual fields, not to mention the convulsions, are enough to distinguish it in most cases. It is often caused by trauma, and frequently figures in court in damage suits, as neurasthenia. There is no doubt, however, that hysteria can coexist with neurasthenia, just as it can coexist with epilepsy.

Hypochondriasis is a state in which there are delusions about the health, rather than real disorders of the health. The hipped state of mind in some neurasthenics suggests a resemblance, but there are not the deep-seated hypochondriacal delusions in the one, nor the genuine sufferings in the other.

In traumatic cases it is important not to overlook organic injury and to call everything neurasthenic. Grave errors have thus been committed.

Secret drug habits, especially the use of morphia, sometimes induce a neurasthenic state, and the true nature of the case may be overlooked. Hence it is important to inquire carefully into the habits. The same is true of alcohol and tobacco, and even of tea and coffee.

Some of the victims of onanism can properly be classed as neurasthenics, but in them there is usually a marked hypochondriacal element. These persons are the easy prey of the advertising charlatans, and their symptoms are sometimes concealed or repressed for fear of exposure.

The Neuroses of War.—The Great War has contributed its share in large measure of cases of the neuroses. Many of these cases answer to the conventional descriptions of neurasthenia, psychasthenia, traumatic neuroses and hysteria. If special types are observed it is because of the severity of the exciting causes, such as "shell-shock," acting on soldiers who are subjected to the fatigues, privations and harassments of war. Shell-shock, by causing violent concussion, may, indeed, cause minute injuries to the central nervous system, which are the foundation of prolonged invalidism and a train of ill-defined but disabling symptoms. To differentiate the "functional" from the "organic" in such cases is often impracticable. On the other hand, severe cases occur in men who have not been injured, and in whom a psychogenic basis is evident. Many cases occur in men who are by nature "neurotic" and have only needed an exciting cause, such as is only too readily found in war.

Many of these war neurotics present well-marked mental symptoms, such as confusion, depression, discouragement and inability to concentrate. Farrar believes that many of these cases are in the main hysterical or present depressive neurasthenic syndromes. Adrian and Yealland point out that the successful treatment is by suggestion and re-education—a fact which tends to prove their purely neurotic type.[1] A well-known English writer confesses that but for his prejudice against the term, he would call most of these cases hysterical.

At the present writing we have not seen many of these cases of the war neuroses in America.

[1] Lancet, June 9, 1917, p. 887.

VI. THE OCCUPATION NEUROSES.

These include a variety of affections which arise from overuse in the course of occupation. They are sometimes called the fatigue neuroses, and are located largely in the neuromuscular apparatus. The commonest forms are scrivener's palsy, telegrapher's cramp, piano-player's hand, and some forms of clergyman's sore throat; and some not so common are seen in fiddlers, bricklayers, and others.

Pathology.—The objection to the term "neuroses" in this connection lies in its implied meaning that the disease is functional. No disease is purely *functional* in the sense that it does not depend on organic change, for there must be some change, however slight and transient. In some of these cases we even see evidences of structural change, such as muscular atrophy, persistent pain, etc.; and we may regard all of them as instances of disordered nutrition both of the nerve-cells and of the muscular attachments. Farther than this it is not possible to go.

Symptoms.—It will be best to consider a few of these affections in regular order.

Scrivener's palsy, or writer's cramp, is marked particularly by spasm, tremor, and incoördination. Pain and vasomotor disorders are sometimes seen. Paralysis and anæsthesia are doubtful symptoms. Three types are noted,—the spasmodic, the paralytic, and the tremulous,—but the distinction is not always clear in practice. The spasm affects chiefly the small muscles engaged in writing, and as a rule is only manifested on attempts at writing—not in other coördinate movements. There are exceptions, however, to this rule. The muscles are held in cramp-like rigidity, wholly preventing the act of writing. In some cases the muscles of the forearm, upper arm, and shoulder are affected, and rare cases are seen in which even distant muscles, as those of the foot, or of the other hand and arm, are involved. The spasm is not a true cramp, for it is non-painful; but exceptions to this rule occur. It is quite involuntary, and may even persist for some moments after the attempt at writing has ceased. Tremor and incoördinate jerky movements are sometimes seen. Paralysis is extremely rare, but a stage of slight paresis may follow the cramp. Pain, or a painful sense of fatigue, is sometimes present, together with painful points on the nerve-trunks. Neuralgic pains are observed in a few cases. Anæsthesia is very rare. It is probably an hysterical symptom. Vasomotor and trophic disorders are not common; turgescence of the limb and flushing of the face are among the former; and such observers as Eulenburg and Gowers claim to have seen muscular atrophy. It has also been seen in a blacksmith, in a saddler, in a tailor, in a dragoon (from holding the reins), in a morocco-worker, and in a player on the bass violin. Atrophy of individual muscles, or small groups of muscles, is seen in some artisans who overuse these muscles, as the small muscles of the hand in locksmiths; and ulnar paralysis and atrophy in glass blowers from pressure rather than overuse.

Telegrapher's cramp is closely allied to the preceding; in fact, it is identical with it except that the cause and the seat of the spasm differ. It was first described by Onimus in 1875 and called by him *mal télégraph-*

ique. Fulton studied the movements engaged in using the Morse instrument, which particular instrument has been held most to blame. The letters are made by a series of dots and strokes which require a fine muscular movement, and when the dispatcher is working rapidly and for long stretches the strain on the muscular apparatus is great. Fulton estimated that an operator makes between thirty and forty thousand contractions per hour. The word "occupation," for instance, contains ten letters and requires twenty-five distinct impressions. One of these patients had worked as long as fourteen hours a day. Another said that his first difficulty arose in making the letter B (— · · ·); his hand would act involuntarily and make the dash and four dots (— · · · ·) which means the numeral 8. Finally his greatest difficulty was experienced in making the letter P (· — — · ·), the rapid succession of dots causing a cramp. In another patient the cramp was painful and was located in the extensors, pulling the hand away from the key (Fig. 404). Worry and discouragement are often seen in these opera-

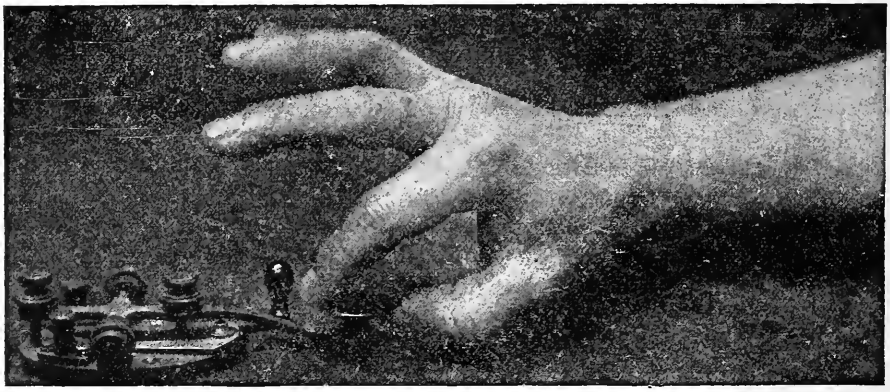


FIG. 404.—Telegrapher's cramp.—Lloyd.

tors; it is easy to make mistakes in transmitting, and these may be serious; consequently a neurasthenic state is often present, which is not improved by alcohol and tobacco.

The telegraph operator can train his other hand rather more easily than the scrivener, because the movements are less complex; but in time the second hand is likely to become affected in both cases. It is easy for the disabled telegrapher to become the victim of writer's cramp.

The *dysphonia* and *aphonia* of professional voice-users have many points of similarity to writer's cramp. There is a spasmodic type and a paralytic type. In singers the voice may break down in the midst of song, although the natural voice for speaking may remain, thus showing how a particular *function* is involved. In the so-called clergyman's sore throat there may really be some pharyngitis or laryngitis, but the nervous element too is usually well marked. There is indeed a well-marked hysterical element in many of these cases of aphonia. Thus Mackenzie reported a case of a fish-hawker who lost his voice suddenly while crying his wares; he was voiceless for four months, and was then cured promptly with electricity.

It is needless to describe in detail the long list of the fatigue neuroses. Among the artisans and artists affected are grinders, blacksmiths, engravers, cigaratte-rollers, pianists, violinists, bricklayers, tailors, and typesetters. Milker's spasm is seen in this country and is said to be common among the cow-herds of the Tyrol. Miner's nystagmus is a curious form.

Diagnosis.—The history and the symptoms are unmistakable. It is always well to recall that a neurasthenic state is often seen in these patients, and that it may complicate the clinical picture. This is still more so of hysteria. Some of the symptoms seen are distinctly hysterical, as, for instance, paralysis and anæsthesia in writer's cramp, for these two symptoms are probably never seen in pure cases. The same is true of aphonia in habitual voice-users; it usually bears all the marks of an hysterical affection. Still, these facts do not necessarily obscure the diagnosis. Writer's cramp has been confused with other diseases in which writing is affected. Among such diseases are hemiplegia with aphasia and agraphia, general paresis, locomotor ataxia, paralysis agitans, disseminated sclerosis, chorea, progressive muscular atrophy, alcoholic and metallic poisoning, and neuritis.

VII. MIGRAINE.

Hemicrania, migraine, or sick headache, is one of the explosive neuroses. Its chief symptoms are disorders of vision, pain in the head, and vomiting. It is paroxysmal, but has no regular periodicity, as a rule, and it is often hereditary or familial. Möbius claimed that 90 per cent. of the cases show heredity.

Etiology.—This is unknown. English physicians are fond of associating "megrim" with gout, and in France it was Trousseau who said, "Migraine and gout are sisters." But these assertions are not proofs. Reflex causes, eye-strain, adenoids, womb disease, and even decayed teeth have been assigned as causes, without much reason. The disease may possibly be due to autoinfection, and gastro-intestinal disorders sometimes excite attacks.

Symptoms.—Migraine usually begins in early life. The initial symptom of the paroxysm is an aura, usually sensory, and in the vast majority of cases visual. The visual auræ consist of attacks of amblyopia in which a part or the whole of the visual fields is obscured; thus scotomata and even hemianopia occur. There are also scintillations, zigzag lines, shaped like a fortification, flashes of light, blazing or flaming splotches, and in very rare cases illusions, as of the forms of animals or terrifying apparitions. Oscillation of the pupil (the so-called *hippus*) is sometimes seen. Other sensory auræ occur, as a numb or tingling feeling in the face, tongue, or one limb. It is characteristic of the aura of migraine to disappear just before the onset of pain.

The pain of migraine is an intense headache. It is usually described as a hemicrania, but it is not always confined to one side. Occasionally, however, it is strictly localized in one branch of the trigeminus, especially the ophthalmic division. The pain increases gradually, until it reaches its acme, and it may endure only for a few hours or even for a day or two.

It is usually terminated by the occurrence of vomiting, which thus constitutes the third or terminal stage of the paroxysm.

Gastric disorder is one of the three characteristic symptoms of migraine. Aversion to food, and even nausea, may occur early in the attack, and sometimes vomiting begins early; but commonly vomiting is a late or terminal symptom. In many cases the patient is promptly relieved by emesis.

Aberrant types of migraine are seen, in which one or other of the cardinal symptoms is wanting. The commonest is the mild form in which the visual aura is followed by slight headache, which is only transient, and the paroxysm aborts without the third stage, or stage of vomiting.

A curious and rare form is the *psychical migraine*, in which mental disorders of various kinds predominate. There is confusion of ideas, with emotional excitement or depression, attending the visual aura, and pain may or may not be marked. Incoherence and even aphasia have been noted, and Liveing attempted to show, with remarkable prescience, that the aphasia always occurs in cases in which the sensory aura, as numbness of the hand, is on the *right* side, thus indicating a *left-sided* cerebral lesion. Genuine *substitutional* attacks have been noted, just as in epilepsy; thus Sir George Airy observed in his own person the attack complicated with transient impairment of speech and memory without either headache or numbness. Tissot observed a case in which attacks of habitual migraine were at length completely replaced by fits of disordered ideation. Hysteria doubtless complicates some cases of migraine.

Ophthalmoplegic migraine is the form in which the paroxysm is complicated with paralysis of some of the ocular muscles. The muscles involved are usually those supplied by the third nerve; but occasionally the fourth or the sixth nerve is affected. The pain is usually severe, and is followed quickly by the paralysis, which may endure for days or even weeks. The bout of pain is commonly terminated by a crisis of vomiting, as in ordinary migraine, but the paralysis constitutes the true terminal stage, and may be total and complete in the third nerve; that is to say, all the muscles supplied by that nerve are involved and are completely paralyzed. There is ptosis, external strabismus, and the pupil does not react to light or on accommodation. In some cases, however, the paralysis is not total or not complete. The duration varies. In Schilling's case the palsy lasted for from four to six weeks, and in Parinaud's case from two to three months; but in many cases the duration is only for a few days. The paralysis, as a rule, to which there are few exceptions, always occurs in the same eye in successive attacks. In some cases the paroxysms observe a true periodicity; in Suckling's case they occurred every two weeks. Many of these cases date from early childhood. Occasionally a permanent palsy results after repeated seizures. Paralysis of the *fourth* nerve has been noted by Leizenberger; and conjoint paralysis of the third and fourth has also been seen. Paralysis of the *sixth* nerve, either alone or in association with some of the fibres of the third nerve, has been reported. De Schweinitz saw a case of abducens palsy, with migraine, which had begun in early life. A very rare case is that of Rossolimo in which a recurring paralysis of the *seventh* or facial nerve

was always ushered in by a migrainous attack. Anæsthesia of the *fifth* nerve has been noted in a very few cases, especially in the first and second divisions, or even in the supra-orbital branch alone. Troemer has reported a case of *ophthalmoplegia interna* following severe attacks of migraine. The pupil was widely dilated and immobile to light, but the other ocular muscles were not affected.

All forms of migraine tend to grow less or even to disappear in middle life.

Diagnosis.—The ordinary migraine is easily recognized; in fact, the patient usually knows well enough himself what he has. The beginning in early life, and the peculiar evolution of the paroxysm, from the aura to the critical vomiting, are unmistakable. Simple neuralgic attacks, which are not uncommon, are known by the absence of the true migrainous symptoms, such as the aura, the explosive onset, and the crisis of vomiting.

Migraine has been likened to epilepsy, especially by Hughlings Jackson and his followers, the resemblance being based largely on the abrupt onset with an aura; but the likeness is superficial. In migraine there are no convulsions, and the disease does not merge into epilepsy. Migraine and epilepsy may, however, coexist in the same person, but the attacks are separate.

Ophthalmoplegic migraine simulates organic disease, especially tumor and syphilis of the brain. The paroxysmal nature of the attack, however, and the tendency for the paralysis of the third nerve to disappear are against brain tumor, as is also the history of the case. In brain tumor, moreover, there are usually other symptoms, such as choked disk and other and more wide-spread paralysis, and the course is progressive.

Syphilitic meningitis between the peduncles causes paralysis of one third nerve, sometimes of both third nerves, and this paralysis may even be evanescent, with severe headache; but the history is not that of migraine, nor is the onset so abrupt, the cure so complete, and the headache so paroxysmal with a crisis of vomiting, as in migraine. Moreover, there are likely to be other nerves involved in syphilis, such as the optic nerves, or even a hemiplegia, and the symptoms are irregular. Opthalmoplegic migraine is always unilateral; syphilis may or may not be so. One of Charcot's cases of migraine had paralysis first of the sixth nerve on one side, then of the third nerve on the other, but the case was unique.

Paroxysmal or recurrent palsy of the oculomotor nerve has been noted also in tubercular meningitis, in otorrhœa, and in nasal catarrh, but the history in these cases and the associated symptoms are against mere migraine.

Some observers claim a relationship between tabes dorsalis and migraine —suggested by the recurrent palsy of the third nerve, sometimes seen in tabes. But it is not scientific to call the recurrent palsy of the third nerve in tabes migrainous; for this palsy is probably due to nuclear disease, and it is to be known by its association with other tabetic symptoms. The same may be said of the association of migraine and general paresis. Syphilis

is the cause of tabes and paresis and can usually be demonstrated by the appropriate tests; not so of migraine.

Hysteria may unquestionably complicate or simulate migraine, especially in the emotional and psychic symptoms, and in the contraction of the visual fields, which might suggest scotoma. But genuine paralysis of the third nerve is probably never seen in hysteria, although it is sometimes simulated by a blepharospasm.

VIII. PARALYSIS AGITANS.

This affection is usually called a disease of old age; nevertheless it occasionally begins in comparatively early middle life; seldom, however, before the age of forty. It is also called shaking palsy, or Parkinson's disease.

Pathology.—The cause and pathology are obscure. Recently C. D. Camp has made an elaborate study of a series of cases, many of them from the Blockley Clinic in Philadelphia, in which he endeavors to show that the essential changes are in the muscular fibres; and he suggests that there may be alteration in the secretion of the parathyroid glands. The ordinary changes of senility, such as atheroma of the cerebral blood-vessels, are commonly found in these patients, but it does not follow, as Dana and a few observers have suggested, that these changes are causative. Some sclerosis of the posterior and lateral tracts of the cord is occasionally observed; also some atrophy of the cerebral convolutions, pigmentation of the motor neurons, and increase of interstitial tissue.

Symptoms.—The disease is of gradual onset and chronic course, and it is rather more common in men than in women. The chief symptoms are tremor, rigidity, paralysis, and affection of the gait. It usually begins with tremor in the hands, sometimes more marked at first in one hand.

The tremor is a regular to and fro or up and down movement, especially marked in the hands, persisting during repose, and temporarily arrested by voluntary motion. The amplitude varies; sometimes it is slight, at others wide and violent, particularly if the patient is aroused or excited. The arrest on volition is but for a moment; the tremor then returns in spite of the patient, and may even be aggravated for a short period. This is seen on attempts at writing. In the lower limbs the tremor is usually not so marked, and is sometimes even absent. It is seen occasionally in the facial muscles, and in the tongue, especially when it is protruded. The statement that the head is not involved is not correct for all cases. Nystagmus is not present. The tremor ceases during sleep.

Rigidity is usually most marked in the hands, arms, back, neck, and face, and its effect is to give the patient a peculiar attitude and expression. Later the lower limbs are involved. The hands are held in the position known as the "obstetric hand," the fingers partly extended, the finger-tips approximated, and the thumb held close. The constant slight movement of the end of the thumb and tips of the fingers gives the appearance in some cases of rolling pills. The arms are generally flexed at the elbows; the back is bent forward, the head is bowed, and the face has a characteristic mask-like expression. In those rare cases in which the tremor is

lacking this mask-like expression and statuesque attitude are enough to establish the diagnosis. The active, intelligent expression of the eyes is sometimes in marked contrast with the expressionless face. The rigidity of the limbs is very noticeable on attempts at passive motion. The speech is often low and feeble but not characteristically changed.

Paresis, or weakness of the muscles, is not uncommon as the case advances; it is doubtless due in part to the embarrassment caused by the tremor and rigidity, but that there is true loss of power there can be no doubt. In advanced cases a genuine paralysis exists, and the patient is confined to a chair, or is even bed-ridden. The knee-jerks may be increased, but there is not usually a true paraplegia, for the bladder is not involved. Sensation is not affected, as a rule; but some patients complain of a subjective sense of heat or cold; and the surface temperature, according to Gowers, may be increased.

Alteration in the gait is shown in the condition known as propulsion or festination. The patient seems to be propelled forward. With head bowed and back bent forward he goes at a shuffling gait or a jog trot, gradually increasing until he brings up against some person or thing which is his objective point. It seems as though he must inevitably fall, which, however, he seldom does. Trousseau said that the patient seemed to be chasing his centre of gravity. Sometimes there is retropulsion; and very rarely lateropulsion.

The mental faculties are not affected except by the changes incident to old age. Parkinson made the curious observation that in a hemiplegic attack the tremor in the paralyzed limb was arrested, sometimes, however, to return. When the disease begins in early middle life the patient often looks prematurely senile. Very rarely the disease is unilateral.

Diagnosis—This is not difficult, for paralysis agitans apes no other malady unless it be some forms of senile tremor; but senile tremor is usually coarser, it is almost always of the intention type, it involves the head; and the characteristic attitude, expression, and gait are wanting. In elderly men, given to the overuse of alcohol and tobacco, a tremor sometimes develops which is not so much senile as toxic. It is worse on voluntary movement, and is not associated with the characteristic symptoms of paralysis agitans. There should be no confusion between Parkinson's disease and multiple sclerosis; the intention tremor, the nystagmus, the scanning speech, the onset in earlier life, are not seen in the former.

There are a few other tremors which it is well to recall in this connection.

A coarse tremor sometimes occurs in the traumatic neuroses and in hysteria. The French describe all these neurotic tremors as hysterical, and they are probably correct. The cases of pseudosclerosis, described by Westphal, were doubtless of the same class. In most of these traumatic and hysteroidal cases the tremor is usually of the intention type, that is, it is worse on voluntary movement, and it is rather coarse; moreover the history and progress are suggestive. These cases often occur in young persons; the attitude, gait and expression are not those of Parkinson's disease; and, finally, hysterical stigmata are often present. In an occasional case the tremor may be fine.

The tremor due to metallic poisoning (by lead and mercury especially) is similar to the hysterical tremor.

The tremor of alcoholism is also largely of the intention type and is coarse and irregular; it is easily known by the history.

X. THE TICS.

The tics, or *maladie des tics,* or habit spasms, are peculiar motor disorders in which one or more groups of muscles are thrown into regular and oft-repeated contractions, resembling a voluntary act. The muscles most frequently affected are those of the eyes, face, mouth, tongue, and neck, but in some cases the muscles of the limbs are involved.

Pathology.—The French, who have described the tics most carefully, point out the underlying mental state. The tic has a quasi-voluntary character, and in its origin it is volitional or impulsive. Gradually the movement becomes fixed as a motor habit, and can no longer be controlled, or only imperfectly controlled, by the patient, and then at the cost of mental distress and anxiety. Usually there is no very distinct motive or associated idea in the mind; the tic is merely an impulse, which grows into a habit. In proof of its psychic character, however, is the fact that in some rare cases the movements are thus associated with definite ideas in the patient's mind, emotional or otherwise, of which the tic is the facial expression; and in extreme cases there may be obsessions, especially of speech, the patient being impelled to give utterance to some set of words, even an indecency—the so-called coprolalia. In fact, the tics are closely allied to the obsessions; they are due to a sort of imperative motor impulse, and they are usually found in neurotic and degenerate patients.

Symptoms.—As no two cases are exactly alike it is difficult to give a brief description of the tics. Blepharospasm is common, and with it is often seen an associated movement of the face and even of the tongue and larynx. Odd grimaces are the result, and these may even be, or seem to be, the expressions of various mental states, as grief, surprise, pain, or joy. When unilateral, as is often the case, the one-sided expression can best be interpreted by covering the sound side of the face. In many cases, however, there is no such expression, but merely a distortion of the features. When the neck muscles are affected there are various movements simulating torticollis. Sometimes associated movements of the arm, hand, leg, or foot are seen.

The tics occur in regularly recurring bouts, the intervals varying from a few minutes to much longer periods. They can sometimes be controlled for a time by an effort of will, but the effort causes mental discomfort, and the patient seeks relief in what Church has called a "spasmodic debauch," in which for a time the tic is repeated rapidly and frequently.

The tics commonly begin in childhood or in young persons. Occasionally they appear later in life; and blepharospasm may have its origin in some affection of the eye, and gradually become fixed as a habit.

Diagnosis.—As already said, the tics are allied to the obsessions and to various neuroses. They are easily recognized by the conspicuous motor disorder; yet they have sometimes been confused with forms of epidemic

hysteria, such as in the "jumpers" of Maine and Canada, and the disease known as "latah" among the Malays, and "myreachit" in Siberia, in which imitation and suggestion are prominent factors. But the ties are not a form of hysteria; they do not occur in epidemics; they are but little influenced by suggestion; and they are too apt to be incurable. It is likewise an error to describe this disease, as some authors do, under the head of chorea. There is nothing choreic about it. In young children these habit spasms in the face, neck, and shoulders, at the very beginning, may suggest St. Vitus's dance, but the tic is more localized, more habitual, and more persistent than the movements in chorea, and there is always a volitional element in it. There is a mild grade of tic in children which tends to recover.

The true ties are to be distinguished from the spasmodic form of tic douloureux, which is a form of facial neuralgia in which the facial muscles are thrown into spasm by the action of pain. In the habit tics pain is absent.

Stammerers sometimes develop a kind of associated spasm in the face or even in a limb. When the embarrassment of speech is great the facial muscles are contorted, and even the hand and arm may be moved spasmodically. This cannot be called a tic in the proper sense of the word.

The ties differ from mere spasm by the psychic element in them. In pure spasm the affection is in the neuromuscular apparatus, and there is no mental collaboration. Such a spasm, purely local, oft repeated, and not involving consciousness, is sometimes seen in some isolated muscle-groups, as, for instance, in the head and neck, constituting the disease known as torticollis. It is usually not controllable by the patient's will, and the cause and pathology are obscure.

VASOMOTOR AND TROPHIC DISEASES.

I. THE AUTONOMIC NEURON SYSTEM. VAGOTONIA AND SYMPATHICOTONIA.

Clinical interest has been much stimulated in the past few years in the sympathetic nervous system and its diseases. Although much of the writing on the subject is theoretical and not a little obscure, nevertheless the importance of the subject must not be ignored. Systems of nerve fibres arising in the central nervous system are distributed to glands outside of that system, and thence these glands send non-medullated fibres to smooth muscles, heart muscle, or secreting structures. As Barker says, "What was formerly known as the sympathetic nervous system is now classified as a part of this great autonomic nervous system, so called on account of its relative independence of the central nervous system."

This autonomic system is very widely distributed, and its disorders must be more or less mingled with disorders of many organs and functions throughout the body. Hence a detailed discussion is not practicable here. The ciliary body and the iris, the heart muscle, the smooth muscles of the blood-vessels and of the respiratory tract, also of the stomach and intestines, of the genito-urinary apparatus, the vessels supplying various viscera, the skin, the mucous membranes and the sweat-glands, all are supplied by these

sympathetic fibres. The viscera are doubly innervated, by a sympathetic system and by a "parasympathetic" system, which are opposed and which maintain a normal balance which may be disturbed in disease. These two antagonistic systems are called the *autonomic nervous system* by Langley.

In the condition known as *vagotonia,* according to Eppinger, Herf, and others, there is hypersensitiveness to pilocarpine, insusceptibility to sympathetic stimuli, and heightened tonus in the parasympathetic system; while in *sympathicotonia* there is hypersensitiveness to epinephrin, relative insusceptibility to pilocarpin and atropin, and heightened tonus in the sympathetic system.

These conceptions, as Barker very truly warns, must not be too rigidly defined. In fact, the present writer believes that the proper interpretation of sympathetic or autonomic syndromes in relation to diseases of the nervous system is a most difficult, and often an impossible, task. It becomes still more difficult when these disorders are looked upon as manifestations of the activities or perversions of the endocrine glandular system. The relation of this whole subject to the psychoses, and psychoneuroses, such as psychasthenia, neurasthenia, hysteria, and the traumatic neuroses, and to such well-recognized clinical forms as Raynaud's disease, erythromelalgia, angioneurotic œdema, etc., is most difficult to interpret and describe. But under these various headings brief references will be found in this volume.

II. RAYNAUD'S DISEASE.

This is a trophic disease in which the extremities, especially the fingers and toes, are the seat of recurring pallor, congestion, and even gangrene. The affection was first described by Raynaud in 1862. It is apparently of vasomotor origin, and has three types or stages—local syncope, local asphyxia, and local gangrene.

Pathology.—The disease seems to be essentially due to vasomotor disorder. Neuritis and arterial sclerosis, or endarteritis obliterans, are not necessary parts of the process, although they may be present as secondary phenomena in late stages. This whole subject of the pathology, however, is still obscure.

Symptoms.—In *local syncope* the parts become blanched, shrivelled and cold; the appearance is similar to that caused by exposure to cold, as in the so-called "dead fingers." The parts affected are usually the fingers and toes, and the affection is symmetrical. The fingers feel numb, and there are anæsthesia and analgesia, but as a rule no loss of the sense of heat and cold. The affection is most common in cold weather, and it is paroxysmal and recurrent, the attacks lasting from a few minutes to several hours. There is no real paralysis, but the fingers may be awkward or even powerless from the numbness and stiffness. There is usually no pain; merely tingling and numbness.

In *local asphyxia* the reverse of the preceding picture is seen; it is a stage of reaction, apparently, although it is not necessarily preceded by a well-marked stage of syncope. The skin becomes dusky, red, purplish, or even almost black; the parts, as the fingers, are congested; the surface is cold; the tactile sense is impaired; and the members may be most painful. The radial pulse remains unchanged. As in syncope, the attacks are

paroxysmal and recurrent, and even occur in cycles. The affected fingers are usually involved in turn, and the congestion disappears first from one part and then another. Occasionally the nose, ears, and face are invaded.

The *local* or *symmetrical gangrene* may follow either the syncopal stage or the asphyxial stage. The parts are cold, usually shrivelled, and bullæ form; these break and reveal black spots, which result in localized destruction. The ends of the fingers are destroyed, or the under parts of the toes. The spots sometimes heal, leaving a healthy scar. The pain is often severe.

Among accessory symptoms should be mentioned hæmoglobinuria, which, being associated with chill and slight fever, and having a paroxysmal course, has led to the suspicion of malaria; an idea which seems to find some favor with Barlow. Amblyopia is also occasionally seen, and may depend on alteration in the calibre of the retinal vessels, as verified

FIG. 405.—Raynaud's Disease.—Dehio.

by Galezowski. Temporary changes in the joints have been noted, also hemiplegia and aphasia, and in rare cases even mental symptoms. Epilepsy, or attacks suspiciously like it, has been reported.

Diagnosis.—There are various forms of gangrene which must not be mistaken for Raynaud's disease. The disorder closely resembles chilblain or frost bite, but the history is sufficient to prevent error. Raynaud's disease is a recurring affection, whereas frost bite is a simple and solitary accident, and by no means always symmetrical.

It also resembles ergotism, but the history alone should distinguish the two.

So also of the gangrene of diabetes, in which the glycosuria and the asymmetrical character of the gangrene are significant.

Erythromelalgia resembles the asphyxial stage of Raynaud's disease, but in the former the affected limb is hot, pulsating, and more uniformly painful than in the latter; moreover, it does not present sensory changes, and gangrene does not result. Nevertheless, the two affections have some affinity. It has even been claimed that they are identical.

Morvan's disease closely simulates the late or gangrenous stage of Raynaud's disease; but the former is closely allied to syringomyelia or central gliomatosis, and there is a peripheral neuritis and thickening of the arterial coats. Hence in Morvan's disease there is usually seen muscular atrophy in the extremities, anæsthesia of all the modes of sensation, changes in the deep reflexes, and possibly scoliosis or kyphosis. The history is not that of recurring paroxysms, and the whitlows are painless.

Injury to a nerve, especially the median nerve, may cause gangrene of the finger-tips, as in a case lately recorded by Sneve, but the history usually is clear, the affection is unilateral, and the paralysis and anæsthesia are characteristic.

Local gangrene may result from the obstruction of an artery, as in the condition called endarteritis obliterans, the pathology of which is obscure.

We do not agree with Oppenheim in confusing the various trophic lesions of tabes, syringomyelia, and even hysteria with those of Raynaud's disease. The associated symptoms of these diseases are sufficient to distinguish them.

Barker has recently called attention to the subject of *acrocyanosis* in which there is anæsthesia with gangrene of the toes. It seems to be a vasomotor affection and may have to be distinguished from Raynaud's disease by the difference in the state of sensation.

III. ERYTHROMELALGIA.

The affection to which Weir Mitchell gave this name in 1878 is described by that author as a chronic disease in which a part or parts of the body, usually one or more extremities, suffer with pain, flushing, and local fever, made far worse if the parts hang down.

Pathology.—The disease has often been ascribed to vasomotor disorder. Lately, however, the tendency has been to attribute it to a peripheral neuritis; although careful examination has not always supported this view. In most cases changes in the blood-vessels, as atheroma and arterial sclerosis, have been present. Finally it has been suggested that there is a central or cord lesion, and cases have been reported by Collier in multiple sclerosis, tabes, and myelitis. It is also seen in hemiplegia. The disease has been observed not infrequently in workmen who do heavy labor and stand for long periods; and Sturgis suggests that it has points of resemblance to the occupation neuroses, such as writer's cramp. On the whole the question of the pathology is still an open one. The weight of evidence seems in favor of a conjoint arterial sclerosis and peripheral neuritis; while a central influence may also act.

Symptoms.—When the limb hangs down it becomes congested and rose-red, or even of a dark violaceous hue; the arteries throb; and the local temperature of the skin rises. Pain is usually present, and may be severe, of a burning or neuralgic kind, worse on pressure, and generally relieved when the limb is again elevated; but sometimes it persists in a minor degree even then. It is worse in summer, and is relieved by the application of cold. Sensation of all kinds is preserved. The disease may

be asymmetrical, and is not followed by gangrene, although a somewhat similar condition has been seen to precede senile gangrene. The symptoms, when in the feet, are usually provoked by walking and standing. It may be questioned whether erythromelalgia is a distinct disease, or anything more than a symptom of various diseases.

Diagnosis.—The distinction from senile gangrene is sufficiently shown in the account of the symptoms. In erythromelalgia the painful swelling is not constant, but is aggravated by the dependent position, and it does not lead to destruction of tissue.

Raynaud's disease is not dependent on position; it begins as an ischæmia; pain is inconstant at first; there is anæsthesia, analgesia, and lowered temperature; and there is a local, and usually a symmetrical, gangrene. But Voorhees, in a recent paper, maintains that the two diseases are merely different phases of the same condition.

The disease has some resemblance to neuritis and injury to the nerves, but there are not the objective symptoms, such as paralysis, anæsthesia, and trophic changes; when these occur it is doubtful whether the case is one of pure erythromelalgia.

It is well to recall, however, that some of the above data for differentiation are not entirely reliable, for pallor, ischæmia, sensory changes, etc., have been noted in cases closely resembling erythromelalgia, and gangrene has even followed in some of them, as in one of Mitchell's cases.

The blue œdema of hysteria is usually confined to one limb; it is likely to be associated with loss of power, and it is often non-painful; moreover, the history and other hysterical symptoms are significant. These hysterical cases usually follow trauma.

IV. ANGIONEUROTIC ŒDEMA.

This is a sort of pseudo-urticaria, occurring usually in weakened or neurotic persons. It is also called giant urticaria, or Quincke's disease.

Pathology.—The affection is probably of vasomotor origin, although the essential cause and its mode of action are obscure. Several instances are given, as by Osler and by Milroy, of hereditary transmission through five and six generations.

Symptoms.—Localized swellings occur on the skin in various regions. These are somewhat like wheals, or hives, but they are painless, and do not itch. They are at first, as a rule, slightly pale, but flushing soon follows. The subcutaneous tissue is involved in the œdema, and sometimes the mucous membranes are invaded. Thus croupy symptoms have been caused, and death has even been ascribed to angioneurotic œdema of the glottis. Gastro-intestinal symptoms, such as colic and vomiting, are sometimes seen.

Cerebral symptoms are caused by these angioneuroses. Thus we sometimes see flushing of the face and head, palpitation, throbbing in the temples, tinnitus, dimness of vision, confusion, and emotional disorder. Such cases are not uncommon in neurasthenic and hysterical patients.

Diagnosis.—The disease looks a little like urticaria, but there is no burning or itching. Some authors, however, incline to identify the two, and claim that itching occurs, but this is doubtful of real cases.

When marked cerebral symptoms occur, as in the cases of "rush of blood to the head," there may be some remote resemblance to epilepsy, but the history of the case and the well-marked vasomotor disorder should prevent error.

Similar phenomena are sometimes seen in old alcoholic cases, and in exophthalmic goitre. Purpura, with urticaria and hæmoglobinuria, is doubtless a different disease.

V. HEMIFACIAL ATROPHY.

This is an affection in which, as the name signifies, atrophic changes on one side of the face, brow, and skull are seen.

Pathology.—Autopsies have been rare; probably the most instructive was one by Mendel, in which he found neuritis of the left trigeminal nerve, most marked in the second division, with atrophy of the descending root and of the *substantia ferruginea*. Homen found a dural tumor compressing the Gasserian ganglion. The disease is a trophic one, probably depending on changes in the fifth nerve, although operations on the Gasserian ganglion have not fully sustained this theory. Some ascribe it to changes in the sympathetic nerves. The subject is still obscure.

Symptoms.—The changes are in the skin, subcutaneous tissue, and bone. The muscles supplied by the seventh nerve escape.

The initial symptom is changed color in a limited area of the skin; this may spread until it involves the entire side of the face. The atrophy of the skin follows, but it may be very slow. Atrophy of the bones is more rare. The color of the hair of the beard, eyebrows, and head is sometimes changed to white. The affected part does not sweat in some cases; and some writers have observed lowered temperature ($\frac{6}{10}$ of a degree). Taste is sometimes involved; hearing seldom; sight never. The facial muscles are not paralyzed, but the masticatory muscles, supplied by the motor branch of the fifth nerve, have been found wasted, according to some authors. Twitching of the facial muscles is sometimes seen.

The above are the essential features of the disease. In some cases trigeminal neuralgia is present, and there may be paræsthesia, as numbness and tingling, but objective anæsthesia is uncommon.

In a few cases hemifacial atrophy has appeared in the chronic insane; and in a few instances a hemilingual atrophy has appeared along with a hemiplegia.

Diagnosis.—This presents no difficulty. The appearance of the atrophied tissue is unmistakable.

In morphœa the bones are not involved; and in scleroderma, according to Duhring, there is an hypertrophy rather than atrophy, and the tissue is hardened.

In Bell's palsy the facial muscles are atrophied, and there is paralysis with reactions of degeneration, but the skin and bones are not affected. Turner says that the faradic excitability of the facial muscles in hemiatrophy is increased and that this arises from lessened resistance owing to the disappearance of the subcutaneous fat. The history in Bell's palsy is usually clear, the affection is acute, and the muscles alone are involved.

Fig. 405*a*.—Facial hemiatrophy.

Fig. 405*b*.—Unilateral hypertrophy of the face.

Fig. 405*c*.—Photograph of cast of the palate in case of Fig. 405*b*, showing the enormous thickening of the bone on the left side.

Atlas of Clinical Medicine (Dr. Byrom Bramwell). By courtesy of the author.

So, too, in progressive muscular atrophy and in muscular dystrophy, when the face is invaded, the muscles alone are involved, the affection is bilateral, and the course and appearance are entirely different.

VI. OSTEITIS DEFORMANS: PAGET'S DISEASE.

Definition.—A rare disease of the bones, characterized by the absorption and new formation of bone tissue, which remains for a time uncalcified and leads to curvatures, over-growth and other deformities of the skeleton.

Up to 1900 only 66 undoubted cases had been reported and in 1902 only 11 cases had been observed in North America.

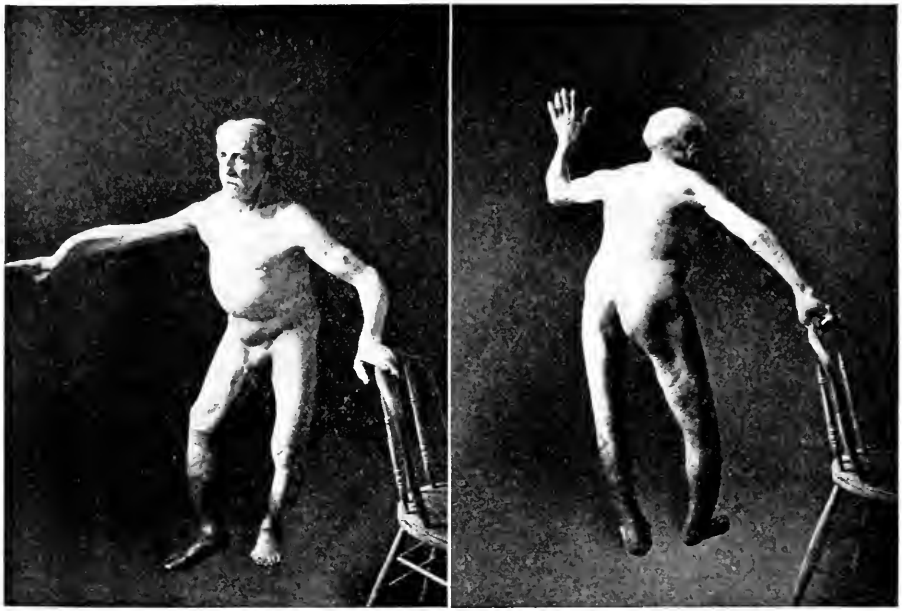


FIG. 406.—Osteitis deformans.—Jefferson Hospital.

Etiology. — PREDISPOSING INFLUENCES. — Both sexes are liable to the disease. Of the reported cases about twice as many occurred in males as in females. Age is more important. The first symptoms have commonly shown themselves after the fortieth year. The onset of the disease in one instance occurred about the age of twenty-one. As the disease is chronic and progressive and in most instances unattended by subjective symptoms, the cases have usually come under observation at a period more or less remote from the time of onset. In the majority of instances the first symptoms have been observed in middle rather than in advanced life. The influence of heredity is uncertain. In three instances, however, cases have occurred in two members of the same family. Occupation is altogether without influence in predisposing to the disease.

Association with Other Diseases.—It has been assumed that there is some causal relation between lesions of the nervous system and osteitis

deformans. No constant relationship has, however, been established and in the greater number of the cases there has been an entire absence of phenomena indicating nervous or visceral disease. Arthritis deformans has in a few instances coexisted with osteitis deformans. This association appears to have been a coincidence and there is no reason to believe that there is any causal relationship or interdependence between the two affections.

The EXCITING CAUSE of the disease remains wholly unknown.

Symptoms.—The manifestations of osteitis deformans are chiefly objective. The onset is insidious, sometimes involving a single bone or a limited number of bones, but in the course of time showing a tendency to symmetrical involvement of the skeleton. Individuals suffering from this disease

FIG. 407*a*.—Skiagram showing deformity of radius and ulna.

FIG. 407*b*.—Skiagram showing deformity of tibia and fibula.

present as the result of definite skeletal deformities a remarkable resemblance to each other. There is thickening of the bones of the skull and an alteration in its shape. The calvarium becomes flattened, the brow broad, the parietal regions prominent. The general circumference is increased so that the patient has to wear a larger hat than formerly. The face is irregularly egg-shaped or triangular, the base being at the forehead and the apex at the chin. The head is carried forward with the chin sunk upon the breast. There is cervicodorsal kyphosis, flattening of the thorax at the upper part, spreading at its base, the abdomen is diamond-shaped and shows a deep transverse sulcus, the hips are increased in width and the lower extremities markedly curved outward and forward, while owing to the decrease in height amounting in some instances to several inches, the arms appear disproportionately long—like those of the anthropoid apes.

Pain in the bones is noted in the early course of many of the cases. In some instances it has been intense. In others it has occurred chiefly at night or after fatigue. As the disease progresses the pains have become less severe. In a large proportion of the cases pain has not been observed. The absence of pain may be explained by the very insidious development of the process.

General muscular atrophy is characteristic of the advanced disease. This is doubtless to some extent due to senile changes in the muscles. There appears, however, to be a definite relationship between the osseous deformities and the muscular atrophy.

Diagnosis.—The direct diagnosis in well-developed cases is unattended by difficulty. The changes in the shape of the head and in the long bones, the diminution in stature, the kyphosis and the peculiar deformities of the thorax and abdomen make up a definite clinical picture not seen in other maladies. The absence of causally related visceral disease and in most instances the absence of the manifestations of lesions of the nervous system and the unimpaired general health are to be noted.

The differential diagnosis involves the consideration of the following diseases:

1. Osteomalacia.—In this affection there is gradual softening and subsequent bending of the bones in which spontaneous fractures frequently occur. There is a feeling of weakness in the lower extremities so that the patient walks with difficulty and requires support. There seems to be some relationship between osteomalacia and osteitis deformans, the essential distinction consisting in the fact that in the latter there is a tendency to the irregular and eccentric formation of new bone.

2. Leontiasis Ossea.—In this rare affection there is hyperostosis of the bones of the skull and face. Osteophytes develop upon the lower jaw and at the margins of the orbits and upon the outer and inner table of the skull. In the latter situation they may cause symptoms of meningitis or tumor. The narrowing of the canals of exit for the cranial nerves may give rise to blindness, deafness, anosmia and peripheral derangements of sensation and loss of motion.

3. Rickets.—This disease of early life presents changes in the bone and other associated symptoms that are characteristic. The bending of the ribs, enlargement of the wrists, squareness of the forehead, open fontanelles are derangements of early developmental processes, not modifications of mature structures. The deformities produced by rickets bear only a superficial resemblance to those caused by osteitis deformans.

4. Acromegaly.—The thick, heavy lips, protruding under jaw and broad deformed face and the enlargement of the head in its anteroposterior diameter bear no resemblance to the cranial and facial changes in osteitis deformans. In acromegaly the bones of the hands and feet and in some instances the epiphyses of the long bones are involved, while in osteitis deformans the changes in the long bones mainly involve the diaphysis, and the bones of the feet commonly escape.

5. Pseudohypertrophic pulmonary osteo-arthropathy—a disease characterized by enlargement and deformity of the fingers, hands, wrists, feet and ankles, occurring in patients suffering from certain chronic

pulmonary affections. This deforming affection of the osseous system bears only a remote resemblance to osteitis deformans. The absence of changes in the cranium, the escape of the shafts of the long bones and the constant presence of intrathoracic lesions constitute points of radical difference.

Prognosis.—Osteitis deformans is slowly progressive and requires a number of years, varying from five to fifteen, to attain its maximum development. It has little influence upon the general health and is not a direct cause of death.

VII. ACHONDROPLASIA: CHONDRODYSTROPHIA FŒTALIS.

Definition.—A form of dwarfism characterized by micromyelia and macrocephalia, the trunk being of about the normal size. This remarkable disease of fetal life was first described by Virchow (1858) who regarded it as a form of fetal or congenital rhachitis. Parrot (1878) suggested the name *achondroplasia*. The most satisfactory account is that of P. Marie (1900).

Etiology.—Predisposing influences and the exciting cause are alike wholly unknown.

Pathology.—There is a dystrophy of the epiphyseal cartilages from the earliest period of osteogenesis. The growth of the bones of the extremities at the epiphyseal cartilages is defective or arrested so that they do not normally increase in length. The bones which develop from a primitive membranous matrix, without passing through the stage of cartilaginous formation, as the clavicle, ribs and certain of the cranial bones, are not involved in the dystrophy, a fact which accounts for the great difference in the development of the extremities and that of the trunk and head. The dystrophy has been ascribed to the action of an unknown toxic agent circulating in the blood to which the epiphyseal cartilage substance is peculiarly obnoxious—perhaps a vitiated internal secretion having to do with the regulation of the normal growth of the body.

Clinical Characteristics.—The disease is essentially an affection of fetal life. By far the greater number of cases die *in utero* or shortly after birth. Most of those who survive die in childhood. A few reach adult life. In a limited number of cases the disease has appeared to commence shortly after birth. These constitute the dwarfs known as micromelic.

The deformities are characteristic. Both the upper and the lower extremities are symmetrically shortened. The arm is shorter than the forearm; the thigh shorter than the leg. The lower limbs are bent, an exaggeration of normal curves. The development of the trunk is about normal. The enlargement of the head is characteristic. It may suggest hydrocephalus. The head is not only large; it is also rounded with exaggerated parietal and frontal bosses. The features are large and coarse, especially the nose, which is depressed at its root and rounded at its point with flaring nostrils. There is lordosis affecting the lumbar vertebræ and in females contraction of the pelvis. The hand is small and square, the fingers short, of about the same length and spreading—a deformity which has been designated the *trident hand*. The scapulæ are short. The muscles are usually well developed. The genital organs are normal. Many of the sub-

jects of this disease are obese. The mental powers are as a rule good; in some of the reported cases defective. Comby thinks it probable that the court dwarfs were achondroplasiacs as they are apt to be very intelligent.

The direct diagnosis of achondroplasia is unattended with difficulty. It is to be differentiated from *rickets*, of which it was at one time regarded as a prenatal type by the skeletal changes which do not involve the trunk and are wholly dissimilar in the extremities and from cretinism, of which it was formerly considered a fetal form by the higher degree of intelligence, the peculiar deformities of the long bones which are shortened and of relatively great thickness in the shaft and by the fact that improvement does not follow the administration of thyroid extract.

XV.

THE DIAGNOSIS OF DISEASES OF THE MUSCLES.

I. MYOSITIS.

Definition.—Inflammation of the skeletal muscles.

Primary inflammation of the muscles may occur as an acute, subacute or chronic disease. The following forms are recognized:

1. Infectious Myositis.—This form is very common in Japan. It may affect one or many muscles. Of 32 cases studied bacteriologically by Miyake, 2 yielded negative results; 27 yielded a pure culture of the staphylococcus pyogenes aureus; 2 the cultures of the albus and aureus and 1 the streptococcus. The onset is sudden. There is fever often high, great depression, induration of the muscles followed by abscesses and, unless the pus is completely evacuated, sepsis.

2. Dermatomyositis.—Many muscles are usually affected. The overlying skin is inflamed and œdematous. The muscles are tender, painful and stiff and upon palpation feel inelastic and dough-like. The affection progressively involves new groups of muscles. Upon post-mortem examination, the muscle substance is firm but fragile and the seat of serous infiltration, fatty degeneration and proliferation of the interstitial connective tissue. This form of myositis resembles trichinosis, from which it cannot be differentiated except by the microscopical examination of a bit of the affected muscle.

3. Neuromyositis.—This name was suggested by Senator for a group of cases characterized by marked disorders of sensation.

4. Polymyositis Hæmorrhagica.—These cases present the clinical phenomena of dermatomyositis but to these are added grave circulatory symptoms due to the implication of the myocardium in the process. The muscles show more or less extensive interstitial hemorrhages. This variety is extremely rare.

5. Myositis Ossificans.—Two forms are described, a local and a progressive. The first affects a limited muscle mass and is stationary. The second develops early in life, advances by irregularly recurring attacks and progressively involves many muscle groups. The number

of boys who suffer from this affection appears to be five times as great as that of girls. It is commonly first recognized in late infancy or childhood. The muscles of the neck, trunk and upper extremities are usually involved, the hands and lower extremities escaping. The masseters are sometimes affected. The other muscles of the face escape. The process by which the ossification occurs consists in cyanotic congestion in a localized area of the tissues which upon pressure are found to be of doughy consistency and very painful. After repeated attacks, bony nodules are found within the muscles at the seat of the trouble and exostoses develop. Impairment of function follows and muscular movements are greatly restricted. The general health is not usually disturbed. There is no fever. Developmental

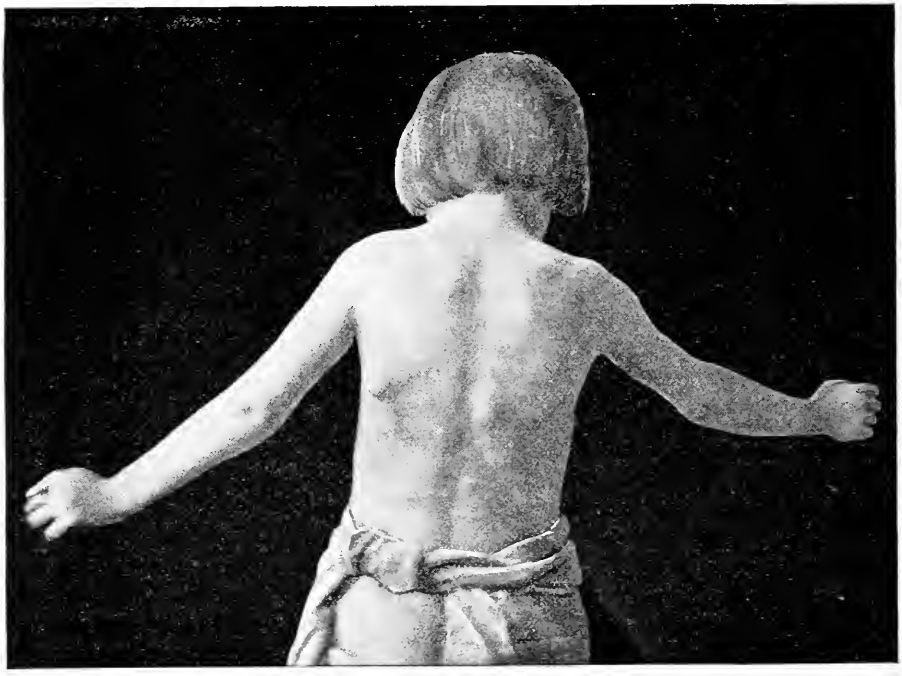


FIG. 408.—Showing exostoses, and full amount of abduction—Walker.

defects are common. They include microdactylia, ankylosis of the interphalangeal articulations, hallus valgus and other deformities of the great toe and malformations of the genital organs. Warren Walker, to whose article I am indebted for some of the above facts, has recently reported a most interesting case. The disease is very rare.

II. THE MYOPATHIES.

By this term we understand essential changes in the muscles, marked by atrophy and loss of power, and not depending on changes in the central or peripheral nervous system.

Pathology.—In the pure myopathies the changes are confined to the muscles. There is atrophy of the muscular fibres, sometimes asso-

ciated with or preceded by swelling in some of them. The nuclei of the fibres may increase, and there is proliferation of the fibrous tissue. Fatty deposits occur, and ultimately the muscular fibres show splitting and longitudinal striations, with the formation of vacuoles. The cause is not definitely known, but there is probably a congenital defect of development or of nutrition of the essential elements of the muscles. As a rule the spinal cord and nerves are normal, although in some cases the motor nerve-endings are involved; and a question arises whether indeed there is not a juvenile type which is not a pure myopathy, but depends upon a peripheral neuritis or nerve dystrophy. The form known as the Charcot-Marie-Tooth type is apparently not a pure myopathy at all, but rather a muscular atrophy dependent upon a degeneration of the peripheral nerves—a so-called primary neurotic atrophy, as shown in autopsies made by Virchow and others. A well-marked type is that in which some of the muscles undergo an increase in bulk, but this is a pseudohypertrophy, in which the overgrowth is not in the muscular elements proper. Various other types of the affection occur, which are rather clinical than anatomical; in fact, these types are probably only varieties of the same degenerative process. It is proper to note that some observers believe that most of these cases are dependent upon a dystrophy of the nerve-endings. The pathology of the myopathies is still somewhat obscure. Recently the theory has been advanced that the myopathies may depend on changes in some of the glands of internal secretion, as, for instance, the pineal gland.

Symptoms.—The onset usually occurs early in life, and may be hereditary or familial. The trunk muscles are affected early in the disease, and the arms and thighs also suffer. There is an absence of fibrillary twitching in the muscles, and the complete reactions of degeneration are not seen. Much refinement of description has been indulged in. The commonest types are as follows:

A juvenile type, or scapulohumeral form, which appears in children, and in which the muscles of the shoulder and arm are first affected.

The facio-scapulo-humeral type, in which, as the name implies, the muscles of the face, shoulder and arm are especially involved. It differs little from the former, chiefly, in fact, in the affection of the face.

The peroneal or leg type, in which the lower extremities, especially the peroneal muscles, are implicated.

Pseudomuscular hypertrophy, in which some of the muscles, especially those of the calf, are enlarged, while others, especially of the back and arms, are atrophied. The enlargement of the calf muscles is not a true hypertrophy, but is due largely to a deposit of fat.

The Charcot-Marie-Tooth type, or primary neuritic atrophy, in which the distal muscles of the arms and legs especially suffer, and in which there is fibrillation along with the atrophy, often with preservation of the knee-jerks, without contractures and occurring as a familial affection. As already said, this type is not a pure myopathy.

The results of these various atrophies, or dystrophies, are various forms of paralysis. Wasting may become extreme, with consequent complete loss of power. The trunk muscles are so affected in some cases that extreme lordosis occurs. When the face is attacked we see the so-called

"myopathic face," in which the oral muscles are especially involved. The muscles of mastication and deglutition, as well as of the eye, are not affected. When the arm and shoulder muscles are involved there results great weakness in the upper extremities, with some deformities of the hands. In the lower limbs the thigh muscles may be so wasted and weakened that the patient cannot stand, much less walk; and in the legs the destruction of the muscles causes various forms of club-foot. In extreme cases no particular "type" is presented, but the patient has extensive atrophy of all the limbs and of many of the trunk muscles, and may become a so-called "living skeleton." In the pseudohypertrophic form the enlarged calves stand out conspicuously, and are hard and brawny to the touch; but the patient usually presents wasting of the arms and trunk. He shows a special difficulty in rising from the floor, climbing with his two hands, as it were, up his legs. In the myopathies there is no involvement of sensation, nor of the bladder or bowel, nor of the mental faculties. The affection is chronic and incurable.

In a recent case of the facio-scapulo-humeral type, observed in the Philadelphia General Hospital, the patient, a young man, had polydactylism of all four extremities.

Diagnosis.—The student or practitioner need not be so much concerned to make out "types" or varieties as to establish a general diagnosis of muscular dystrophy. For this purpose the history of the case is first considered, and then the peculiar atrophy, associated perhaps in some muscles with pseudohypertrophy, without fibrillation, reactions of degeneration, sensory changes or incontinence. These points, some or all, serve to distinguish the affection from the progressive muscular atrophy of adults, or from acute multiple neuritis, or from myelitis as the case may be. From infantile paralysis or acute anterior poliomyelitis the disease is distinguished by the slow onset, its progressive character and wide extent, the preservation of the electrical responses to an advanced stage, and the general history.

The primary neuritic atrophy of Charcot, Marie, and Tooth presents some special points which give it a place almost unique. It somewhat resembles progressive muscular atrophy, also multiple neuritis. It is distinguished, however, by being often a familial affection, beginning rather early in life, very chronic, and the wasted muscles are the distal groups of the extremities. Fibrillation is common and foot-drop is seen. The knee-jerks may be preserved, and sensory symptoms are wanting. A posterior sclerosis of the spinal cord has been seen in some cases, as recently pointed out by George Wilson, giving these cases a resemblance to Friedreich's ataxia.

In the cerebral palsies of children we see hemiplegia, diplegia, paraplegia and rarely, monoplegia. The paralysis is spastic, with exaggerated tendon reflexes and without true muscular atrophy; and in some cases there are cerebral symptoms, as epilepsy and various grades of idiocy.

III. THOMSEN'S DISEASE: MYOTONIA.

This is a bizarre affection, seen by but few persons, and described by most writers in terms exactly alike. It was first reported by a Dr. Thomsen, for whom it is named, and in whose family it seems to have

prevailed to an unprecedented extent. As it is claimed to be congenital, hereditary, and familial, it is sometimes called *myotonia congenita*.

Pathology.— The disease has no pathology that anyone has yet discovered. But little significance is to be attached to such slight changes as are reported by Erb, Dejerine, and a few others, and which seem to consist in nothing but a little increase in size of the muscle fibres.

Symptoms.—Muscular rigidity, or cramp, occurring on voluntary movements or attempts at such movements, is the chief symptom. A condition of tonic spasm sets in when the patient, especially after a long rest, attempts to use certain muscles or groups of muscles. These spasms do not seem painful; at least, pain is not insisted on by some writers. In some cases a few muscles only are involved; in others almost the whole musculature is thrown into tonic spasms. There is no loss of consciousness.

Some effort has been made to show that the muscles present a special or so-called "myotonic reaction" to electricity. This consists in a state, more or less transient, of slight contracture and tonic spasm, which varies somewhat with the kind of current used and with its strength. With faradism there is a tonic contracture of long duration; with galvanism only labile currents produce contractures, which are sluggish in character. There is increase of mechanical irritability, shown by the muscle giving a sluggish tonic contraction on percussion, especially at the point struck.

Diagnosis.— This is not difficult, for the disease is like none other. An attempt should always be made to exclude hysteria.

IV. PARAMYOCLONUS MULTIPLEX.

Friedreich, in 1881, described a disease which has been given this cumbersome name. Few seem to have seen it, or anything like it, and some good authorities deny its existence. It has no recognized cause or morbid anatomy.

Symptoms.—As described by most writers, the disease is manifested by a series of short, quick, irregular, shock-like contractions of the muscles of the extremities and trunk. The face usually escapes. The contractions are not unlike those caused by electric shocks. The intelligence, the sensory system, and the sphincters are not involved.

Diagnosis.—Hysteria is not to be ignored. Lloyd refers to aggravated cases of hysterical tremor, with coarse irregular jerkings, which suggested to him the picture of paramyoclonus as drawn in some books. These are probably the patients who get well.

The *chorea electrica* of Bergeron, Henoch and others is probably a true chorea, although cases of it have been described as paramyoclonus multiplex. Dubini's chorea, also called electrical, is very rare, and is said to be associated with fever, muscular atrophy, and paralysis. It is an infectious disease, seen mostly in Italy.

V. MYASTHENIA GRAVIS.

The disease to which Jolly gave this name may be said to be still on trial. Both clinically and pathologically it is as yet a subject of dispute. It may be defined as a syndrome in which there is rapid exhaustion of the muscles supplied by the bulb, as well as of some of the eye muscles and muscles

of the extremities. The tendency is towards a fatal termination. The disease is also called *asthenic bulbar palsy.*

Pathology.—Almost by unanimous consent the Germans, who have done most to secure the recognition of this syndrome, declare that the anatomical examinations in the brain and cord are negative. Maier claimed to have found alterations in the anterior roots of the spinal nerves, and Marinesco and Widal reported changes in the ganglion cells, but Oppenheim denies the validity of these findings. In America, studies have been made by Hun, Burr and McCarthy, and others, and there is a disposition to see changes in the thymus gland and lymphoid infiltration in the muscles as the true causes of the disease. The subject is still far from settled. E. T. Bell has recently described a tumor of the thymus in a case of myasthenia gravis; and some abnormality of the gland is said to be found in nearly one-half of the cases of this disease.

Symptoms.—Rapid exhaustion of certain muscles or muscle groups seems to be the cardinal symptom. The patient may begin using the muscles with normal vigor, but they rapidly exhaust, sometimes with alarming results. Thus there occur dysarthria, dysphonia, dysphagia, dyspnœa, ptosis, in short, the evidences of paralysis of muscles supplied by the 3d, 5th, 7th, 10th, and 12th nerves. Along with this the extremities are often involved, and the respiratory muscles weakened, so that the prostration may be extreme and dangerous. These attacks may be paroxysmal, excited especially by voluntary use, and in fact the progress of the disease is often by stages.

Jolly observed the "myasthenic reaction" to faradic stimulation, *i.e.*, the rapid exhaustion of the muscles by the faradic current. This reaction is claimed by some to be pathognomonic. There is no muscular atrophy; no fibrillation; no involvement of sensation; no optic atrophy; no paralysis of the bladder or bowel; no abolition of the tendon reflexes (as a rule); no affection of consciousness. Hun's patient, however, had attacks of extreme weakness, or collapse, in which the mind was not clear and the heart's action was depressed. The attacks resembled *angina sine dolore,* or even heart block.

Cases vary in the distribution of the symptoms. In some patients bulbar symptoms are most marked; in others exhaustion is more wide-spread. Some cases continue for years, and the disease may have marked remissions and exacerbations. A fatal result is to be apprehended.

Diagnosis.—The disease resembles Landry's paralysis, except in its tendency to remission and in the absence of evidence of organic central disease. It is probable, however, that some reported cases are allied to, if not identical with, Landry's disease.

In acute anterior poliomyelitis the evidence of organic central disease is clear; as, for instance, a flaccid paralysis with loss of the tendon reflexes and with the reactions of degeneration. The disease is ushered in as an acute febrile affection; it is not one of remissions and exacerbations, and the paralysis is obstinately located, as a rule, in one limb and even in one set of muscles. Bulbar symptoms are not common, although not unnoted, especially in epidemic anterior poliomyelitis.

From true bulbar palsy the asthenic form is distinguished especially by the history and the absence of evidence of nuclear disease. In the former

there is an insidious onset, a slow course, and the affected muscles slowly waste, and present fibrillation and gradual loss of power.

Hysteria may closely simulate myasthenia gravis. The exhaustion and the myasthenic reaction to faradism should distinguish the two affections. Bulbar symptoms are rare, although there may be aphonia, globus, œsophagismus, and retching. Hysterical ptosis is not unheard of.

It is necessary to utter a warning against placing too much confidence in the exhaustion symptom. Something very like it can be seen in neurasthenia; and, in fact, a weakened muscle from whatever cause (as in neuritis or dystrophy) exhausts rapidly on being used.[1]

[1] See Campbell and Bramwell for a critical digest of myasthenia gravis, Brain, 1900.

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